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MINERALS IN POULTRY NUTRITION[1]

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INTRODUCTION

The mineral, inorganic, or ash content of a feed or a bird is represented by the material remaining after incineration of the substance. The last two names are somewhat misleading, since only a part of these minerals is present in the ration or in the bird's body as inorganic elements; the remainder is present in organic combination. For example, sulphur is a constituent of some proteins, and phosphorus is present not only in inorganic form but also in organic combination as nucleoproteins, phosphoproteins, phospholipids and phytates.

Since the greater portion of the mineral content of the bird is contained in the skeleton, the mineral requirements of birds, particularly those of young growing chicks, are often considered on the basis of bone formation only, the fact that minerals have other functions in the living organisms often being obscured. As calcium, phosphorus and, to a less degree, magnesium, play important rôles in this connection, the influence of these minerals on bone formation has been extensively studied. However, they too have other important functions, and interrelationships.

The special functions of some of these minerals will be discussed later. The general functions of mineral elements are as follows:—

1. Minerals maintain the necessary osmotic pressure, surface tension and so forth in the various fluids of the body and so aid in absorption, excretion and secretion.
2. Minerals are concerned in the regulation of the hydrogen ion concentration of the blood and tissues. For example, the acidity of the gastric juice of the proventriculus, which is necessary for the activity of the proteolytic enzyme pepsin, is due to hydrochloric acid derived chiefly from sodium chloride in the blood. The normal pH of blood is partially maintained by the carbonates and phosphates of sodium.
3. The irritability or ability to respond to stimuli of muscle and nerve is dependent upon and influenced by the presence of certain inorganic substances. Calcium is necessary for the transfer of nervous impulses. In this connection there is an interrelationship and balance between sodium, potassium, calcium and magnesium. By upsetting this balance (for instance, by injection of mag-

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nesium sulphate) coma, anaesthesia and paralysis can be induced. The symptoms are rapidly alleviated by the injection of calcium chloride. On the other hand, deficient amounts of calcium in the blood (hypocalcaemia) lead to hyperexcitability and tetany. The rhythmicity of the heart beat is also dependent upon a proper balance between mineral elements. If a beating heart is perfused with a solution, of the proper concentration, of sodium chloride, it soon stops beating. If calcium is added to the solution, the heart resumes beating but it fails to relax properly and finally stops in a state of contraction. Subsequent addition of potassium results in normal beating. But a proper proportion between calcium and potassium is necessary, excess potassium resulting in failure of the heart to contract properly. The chick embryo heart has been utilized by Murray (284, 285) in studying this problem.

4. Minerals are integral parts of living protoplasm. As has been pointed out, phosphorus is an essential mineral in all cell nuclei (nucleoprotein) and in brain and nerve tissue (phospholipins).
5. Minerals constitute the greater part of bones, giving rigidity to the skeleton.

These examples will suffice to demonstrate that minerals are extremely important to the physiological activities of any living organism, in addition to furnishing the framework of that organism.

The question of the "availability" of minerals is important, but the phrases "availability of minerals in a ration" or "percentage absorption of minerals" mean very little since so many factors enter into the processes of absorption and utilization. The "availability" of a mineral is partially dependent upon its ease of absorption which includes solubility. But absorption and assimilation are conditioned by other factors than the "form" in which the mineral is supplied in the ration. Vitamin D increases calcium and phosphorus retention due to either increased absorption from the alimentary tract or lessened excretion from the body or both. Moreover, there is ample evidence that the absorption of an extra amount of potassium involves a greater requirement for sodium. Digestibility trials, even when the technical difficulties attendant upon such experiments with poultry are overcome, give no measure of the utilization of the mineral constituents of a diet, for a mineral may be absorbed from one part of the gut and excreted lower down after fulfilling any function for which it is required while in the bird's body. Iron, if in a soluble form, is absorbed from the small intestine and is excreted into the large intestine. Hence, according to a digestibility trial, iron compounds are not digestible. The difference between the chemical analysis of the ration and that of the combined excreta (faeces and urine) will show the "retention" of any mineral at that particular time and under the particular physiological status of the bird at that time. Previous to the onset of egg production, for instance, the percentage retention of calcium may rise from 20% to 70%.

The minerals taken into a bird's body are constantly being excreted by the kidneys (urine), and intestinal wall (faeces). An excess of a mineral, if within a reasonable range, is rapidly excreted. If a deficiency exists,

the body conserves its supply and lowers its normal excretion. Thus, if common salt is given in somewhat excess amounts, the chlorides in the urine increase. If no salt is fed the amount in the urine rapidly decreases. It will be seen that the kidneys are able to safeguard the body against an imbalance of minerals by this regulatory mechanism but, as will be shown later, if excessive amounts of certain minerals are continuously fed, the safety mechanisms break down, the bird is no longer able to maintain the normal composition of the body fluids and tissues, and injury or death ensues. In fact, the writer is of the opinion that under modern feeding conditions, poultry are more liable to suffer from an excess rather than a deficiency of minerals. However, it should be evident that the percentage retention of any mineral will depend also upon the amount which is fed as well as the previous nutritive regime of the bird to which it is fed. In other words, the availability of a mineral constituent of a ration will vary according to circumstances. For practical purposes, although there is little or no evidence available, it probably is safe to assume that one-third to one-half of the mineral content of a ration is available.

Three very excellent reviews of minerals in poultry nutrition have been prepared by Emslie (102), Cruickshank (82) and Halnan (129). The author has drawn heavily on these publications in preparing this present review. Mitchell and McClure (280) have prepared an outstanding monograph on mineral nutrition. The mineral requirements of poultry have also been discussed by Jull (195), Kaupp (201a), Kennard and Bethke (205a), Halnan (128a), Prentice (321a) and Muller-Lenhartz and von Wendt (282a).

Acid-base balance as influenced by feeding has been investigated by Burchardt (51a), Kaupp and Ivey (201b), Solun and Schuster (356a), Schmidt (339) and Solun and Troitskaya (356b).

1. MINERAL CONTENT OF POULTRY AND EGGS

An adequate mineral supply is necessary for poultry during most of their life span. Their growth is rapid and hence the requirements, particularly of calcium and phosphorus for bone formation, are high. At maturity, egg production necessitates a considerable quantity for the formation of eggs, particularly calcium for shell formation. Halnan (129) gives the analysis in Table 1 for an average egg (including shell) weighing 2 ounces. Mitchell, Card and Hamilton (276, 278), and Halnan (129) have carried out slaughter tests and chemical analyses of different breeds of poultry at various ages. The percentage of ash in the bird's body rises rapidly during the first seven weeks, and then shows a gradual increase to 40 or 45 weeks. In the case of the pullet, a small decrease occurs at the onset of egg production, probably due to the extra requirement and mobilization for egg production. The percentage ash of cockerels is generally higher than that of pullets. It also appears that the percentage ash of heavy breeds (meat production) is lower than that of light breeds (egg

TABLE 1.—MINERALS IN AN AVERAGE EGG*

	Grams.
Calcium	1.975
Magnesium	0.027
Potassium	0.067
Sodium	0.073
Phosphorus	0.115
Sulphur	0.114
Chlorine	0.088
Iron	0.001

* Data of Halnan.

production) at the same age, indicating that "meat" breeds carry a higher proportion of meat to bone than the "egg" breeds. The distribution of minerals in various parts of the hen's body has been studied by Bernardi and Schwarz (14, 15).

Halnan's detailed analysis of 9-months old Light Sussex is shown in Table 2. Kaupp (201a) gave the mineral content of chicks at various ages.

TABLE 2.—COMPOSITION OF FAT-FREE, DRY MATTER*

	Cockerel		Pullet	
	Flesh and offal	Bones	Flesh and offal	Bones
	%	%	%	%
Ash	5.50	38.88	4.98	40.50
	grams	grams	grams	grams
Ash	17.640	68.740	14.220	55.890
Phosphorus	2.848	11.600	2.418	9.880
Sulphur	3.733	0.922	3.160	0.742
Calcium	0.430	25.680	0.345	20.770
Magnesium	0.478	0.807	0.374	0.624
Potassium	4.778	0.787	3.630	0.566
Sodium	1.443	0.713	1.113	0.595
Chlorine	2.283	0.817	1.996	0.552
Iron	0.131	0.030	0.128	0.034

* Data of Halnan.

The distribution of minerals during embryonic development has been studied by Mankin (249), Horning and Scott (176), and by Leubir and Poulant (235).

Spectrographic analyses of eggs and tissues were carried out by Drea (416), showing that a number of elements pass from the feed into the blood, from there into the egg and finally into the chick's tissues and blood. These were aluminium, barium, calcium, copper, iron, magnesium, phosphorus, potassium, rubidium, silicon, sodium, strontium, titanium, and vanadium. Manganese and zinc though present in the hen's ration were absent from the chick's blood, but present in all organs but one.

Of the "trace" elements, barium, iron, strontium and vanadium were more concentrated in the blood and/or egg than in the feed, indicating some physiological importance. Boron was found only in egg yolk, silver was present in the egg, but fluorine was only found in one femur. Chromium, lead and molybdenum were present in the feed and in the hen's blood, but were not constantly present in the eggs.

In a similar study of eggs made in this laboratory through the co-operation of the Hospital for Sick Children, Toronto, all eggs were found to contain large amounts of sodium, calcium, magnesium, potassium and phosphorus, appreciable amounts of iron and silver, and traces of manganese, lead, tin, rubidium, strontium and chromium. Eggs from hens fed on the usual feedstuffs and those fed on a simplified diet including grass ash contained traces of aluminium, whereas without grass ash none of this

element could be detected. The "grass ash" eggs also contained cadmium barium and caesium.

Periodic analyses of bone in normal and abnormal chicks have been made by Russell and Massengale (331), Miller, Dutcher and Knandel (273), Harshaw, Fritz and Titus (139), and Elvehjem and Kline (100). The percentage of bone ash increases from 30 to 36% during the first week, and, in the presence of ample vitamin D continues to increase to 40 to 42% at six weeks. It would appear that these figures for the average ash content of chick bones should not be considered too seriously since the tentative A.O.A.C. method for the bioassay of vitamin D carriers for poultry feeding requires, for satisfactory calcification, an average bone ash content exceeding 45% at four weeks. On a rachitogenic diet the ash content drops to 27 to 29% at five weeks. According to Holmes, Pigott and Moore (173), using Rhode Island Red chicks, and Schroeder (341), using White Leghorns, there is a sex difference in the bone calcification, the bones of females having a higher ash content than those of males. Results of analyses in our laboratory with Barred Plymouth Rock chicks carried on for some years show the reverse, the bones of the males having the higher ash content. Whether this is due to breed difference is an open question. The procedure used also may be responsible. The procedure in analyzing bones has been discussed by St. John, Kempf and Bond (362), Lachat (222), and Bethke and Record (22). According to Lachat (220), this sex difference does not appear on vitamin D deficient diets. Later, Lachat (223), and Lachat and Halvarson (224) reported that there was an association between body weight and bone ash content. On the same ration, faster growing birds not only develop greater amounts of skeleton than slower growing birds, but the relative amount of minerals deposited in the skeleton is greater for the former. This finding is contrary to the conclusion of Harshaw, Fritz and Titus (139) that the percentage of ash in the bones of slower-growing birds is greater than that in the bones of faster-growing birds, whose rate of gain is normal. Limited data from our laboratory would support the latter conclusions. It is obvious that there is some complicating factor which, as yet, has not been controlled. These findings, emphasize the necessity of further research on this fundamental problem. If abnormal bone formation is to be studied with any reasonable surety of success, it is imperative to have some general agreement as to what is normal bone development. In the author's opinion, until such time as it is generally realized that the first four weeks are an insignificant period for such studies, little real progress will be made. Many skeletal changes occur long after this period. He further questions whether four-week assays are fundamentally sound.

Lachat and Halvarson (224) concluded that vitamin D deficiency more seriously affects growth and calcification in females than in males, even though the former utilize the vitamin more efficiently, and reversal of normal sex differences in calcification may occur. The works of Buckner and Martin (35), Buckner, Martin and Insko (37), Wilgus (404) and Holmes, Pigott and Campbell (170) show that calcium and phosphorus are deposited in a fairly constant ratio (approximately 2 to 1) regardless of the completeness of bone development. Pearl (310) has reported a sex differentiation in the effect of calcium salts, using calcium lactate and

calcium lactophosphate. Van der Hoorn and Branion (384, 385) were able to cause a stimulation in the growth of females by the use of grass ash.

In the author's laboratory, in collaboration with H. LeMasurier of Toronto, a roentgen ray study of the skeletal development of various breeds of poultry at weekly intervals during the growing period up to the onset of egg production is being carried on. The results, although by no means complete, show that this field is very fruitful and indicate clearly that our knowledge of avian bone development is rather fragmentary.

In normal chicks the level of serum Ca is approximately 10–12 mg. per 100 cc. and inorganic P about 4–6 mg. However, various workers have reported widely varying values for the inorganic blood phosphorus. These differences can be accounted for, at least in part, by different analytical technique; by differences in the calcium and phosphorus content of the ration, cf. Massengale and Nussmeier (255), Heller, Hunter and Thompson (152) and Heller, Zimmerman and Thompson (157); and by variations in the source and amount of the antirachitic vitamin, cf. Steenbock, Hart, Jones and Black (358), Ackerson, Blish and Mussehl (1), Hughes and Titus (178), and Murphy, Hunter and Knandel (286), and numerous other workers. In this connection, it should be pointed out that Bills, Massengale, McDonald and Wirick (23), have shown that the vitamin D of irradiated ergosterol raises the calcium and phosphorus of chicken serum more effectively than it promotes calcification, whereas the vitamin D of cod liver oil promotes calcification more than it elevates the serum calcium and phosphorus. The age of the chicken is also a factor. Elvehjem and Kline (100), Hall and King (125), Harshaw, Fritz and Titus (139), and Greenberg, Larson, Pearson and Burmester (120) have made weekly blood analyses for calcium and phosphorus. The blood calcium may drop slightly from a level of 13–14 mg. per 100 cc. at two weeks to 10–11 mg. at six weeks, although all workers are not agreed on this point. The inorganic phosphorus level progressively decreases with increasing age. Unfortunately the results of blood analyses for calcium and phosphorus are not comparable since determinations have been made on whole blood, some on plasma, and others on serum.

Although the chicken is very resistant to hypervitaminosis D, excessive amounts do produce some changes in young birds. Hall and King (125), and Steenbock, Kletzien and Halpin (359) have observed a rise in serum calcium. Similar findings were obtained by Massengale and Nussmeier (255) with a ration containing only 0.1% of calcium. Hall and King (125, 126) found no deviation from normal in the percentage of ash and calcium in the bones, but histological examination showed imperfect calcification which was less marked when the irradiated ergosterol was fed with calcium phosphate. The inorganic phosphorus may decrease or show only slight changes.

Calcium exists in the blood in various forms. Presumably the inorganic calcium of the serum measures practically all the calcium of the blood, since the cells are almost devoid of calcium and practically all forms are transformed into and measured by the usual methods for inorganic calcium. The inorganic calcium can be subdivided into diffusible and non-diffusible. The diffusible fraction exists as readily ionizable salts, while the remainder is bound to protein. Some workers prefer to partition

the blood calcium into (1) an absorbable diffusible fraction, (2) a non-absorbable diffusible, (3) a non-diffusible absorbable, and (4) a non-diffusible, non-absorbable fraction. Other workers use the term "filtrable" in place of "diffusible".

Phosphorus is present, not only in the plasma but also in the cells, and owing to the fact that the red corpuscles of birds are nucleated, with resulting nucleoproteins containing phosphorus, the total phosphorus of fowl's blood is 3–4 times that of mammalian blood. Inorganic phosphorus constitutes but a small fraction of the total phosphorus. The phosphorus compounds of the blood can be subdivided into two main groups: (1) acid-soluble which includes inorganic, nucleoproteins, nucleotides and phosphate esters, and (2) alcohol-ether soluble or lipid-phosphorus.

Variations in these different fractions are not proportional as shown in Table 3 and as will be pointed out later in relation to ovulation. It seems possible that determinations of inorganic blood calcium and phosphorus which disregard these other fractions, have retarded the disclosure of more fundamental facts with regard to the influence of calcium and phosphorus on metabolism in poultry.

TABLE 3.—PHOSPHORUS DISTRIBUTION (MG. PER 100 CC.)*

—	Total P	Cell P				Plasma P			
		Total	Lipid	Inorganic	Acid soluble	Total	Lipid	Inorganic	Acid soluble
Normal	115.3	100.0	6.4	1.9	25.0	15.3	9.1	5.9	4.5
Osteoporotic	119.6	106.8	6.3	2.9	27.8	12.9	6.9	4.9	5.4

* Data of Heller, Paul and Thompson.

It is not easy to decide whether a disease, particularly abnormal bone development, is due to a mineral deficiency in the ration, or whether it is due to some fault in the physiological mechanisms for utilizing the mineral; the same pathological picture may result from apparently different causes, while different diseases may appear to arise from the same cause. Three types of disease must be distinguished due to (1) a deficiency of minerals in the diet—acalcicosis (calcium-deficiency), aphosphorosis (phosphorus-deficiency), etc.; (2) a deficiency in factors influencing mineral metabolism in the diet—avitaminosis etc.; and (3) a failure in the physiological mechanisms in the body of the bird which affect mineral metabolism.

It should also be pointed out that low mineral content of the blood may be associated with low mineral content of the ration but there is no necessary connection in some instances. The level of serum calcium is under the influence of a rigorous physiological controlling mechanism, the parathyroid glands, which produce a hormone, operating in association with the nervous system, and co-operating with the organs of storage, absorption and excretion (bone, intestines and kidneys), which, unless the regulatory mechanism breaks down (Hutt and Boyd, 186), maintain the physiologically normal level of calcium in the blood. The level of plasma phosphate is not under such control, or at least, not to the same extent.

Although hypophosphataemia (low blood phosphorus) is characteristic of either phosphorus deficiency in the ration or failure of the bird to absorb and utilize the phosphorus of the ration, hypocalcaemia does not always indicate a calcium deficiency.

Moreover, it seems evident that the time has been reached in a study of the mineral requirements of poultry, when the co-operation of the physiologist, particularly in relation to hormones such as parathormone, but also those hormones which influence egg production, and of the histologist, in relation to bone studies, must be sought. It is extremely doubtful if our present "standard" diets for the production of rickets in chicks produce rickets. True rickets is a phosphorus deficiency disease—be that deficiency produced as it may. Most, if not all, chicks suffering from experimentally produced "rickets", show deviations from normal in both blood calcium and phosphorus. Rickets should result in hypophosphataemia with normal blood calcium. As McGowan and Emslie (264) have pointed out there is no definite proof in the literature of the production of rickets, the evidence indicating osteoporosis. From a practical viewpoint this may not be important, since vitamin D therapy apparently prevents both conditions, but from fundamental considerations, a morphological study of the changes in bone seems essential. In this connection it might be noted that Oberling and Guerin (294, 295, 296) have described lesions of osteitis fibrosa in chicks.

2. MINERAL CONTENT OF FEEDINGSTUFFS

The extent to which ordinary feedingstuffs furnish minerals can be judged from the following table showing the average mineral content of the common poultry feeds gleaned from various sources. It should be clearly borne in mind, in using an average figure for the mineral content of a feed, that the amount of these minerals in any particular lot of feed may differ considerably from the average. The composition of feedingstuffs is greatly influenced by the source and method of processing, by the stage of maturity, and by the water content and amount of plant food, especially nitrogen, calcium, phosphorus and potash in the soil.

TABLE 4.—AVERAGE PER CENT MINERAL CONTENT OF FEEDS

Feed	Ash	Calcium	Phosphorus	Magnesium	Sodium	Potassium	Chlorine
Alfalfa meal	7.6	1.23	0.20	0.40	0.45	1.91	0.15
Alfalfa leaf meal	11.1	2.23	0.30	—	0.11	2.04	0.54
Barley	2.8	0.06	0.37	0.15	0.08	0.49	0.49
Blood meal	3.4	0.28	0.22	0.06	0.48	0.10	0.50
Bone meal (steamed)	80.2	30.26	14.18	0.81	0.48	0.15	0.06
Bone meal (raw)	61.8	23.83	11.00	—	—	—	—
Buckwheat	2.0	0.04	0.29	—	—	0.27	—
Buttermilk (dried)	10.1	1.34	0.81	0.14	0.51	0.71	1.21
Cod liver meal	3.1	0.13	0.75	—	—	—	—
Corn	1.3	0.01	0.27	0.12	0.02	0.32	0.04
Corn gluten feed	4.9	0.18	0.69	0.43	1.03	1.13	0.22
Crab meal	39.5	13.06	0.51	—	—	1.90	—
Distiller's corn grains (dried)	2.1	0.05	0.31	0.05	0.15	0.01	0.07
Distiller's rye grains (dried)	3.3	0.13	0.44	0.20	0.08	0.04	0.03

TABLE 4.—AVERAGE PER CENT MINERAL CONTENT OF FEEDS—*Concluded*

Feed	Ash	Calcium	Phosphorus	Magnesium	Sodium	Potassium	Chlorine
Fish meal (65–70% protein)	14.8	4.08	2.85	0.67	0.40	—	0.60
Fish meal (60–65% protein)	21.0	7.19	3.79	0.21	1.49	0.92	1.35
Fish meal (55–60% protein)	22.6	7.32	3.89	0.22	1.58	0.32	1.65
Fish meal (herring)	14.2	4.32	2.85	0.21	0.74	0.49	0.47
Fish meal (pilchard)	16.5	8.34	4.54	0.28	0.65	0.69	0.42
Hominy	1.6	0.02	0.32	0.02	0.13	0.36	0.06
Limestone	—	38.97	—	—	—	—	—
Linseed meal (old process)	5.6	0.38	0.88	0.58	1.04	1.32	0.44
Linseed meal (new process)	5.6	0.36	0.71	—	—	—	—
Meat meal (60% protein)	17.2	5.42	2.85	—	—	0.55	—
Meat meal (55% protein)	23.9	8.18	4.09	1.17	0.60	1.43	0.46
Meat meal (50% protein)	26.9	10.96	5.16	—	—	—	—
Oats	3.4	0.10	0.39	0.16	0.16	0.46	0.07
Oat middlings	4.1	—	0.56	—	—	0.57	—
Oyster shell	—	37.95	—	—	—	—	—
Rye	2.0	0.05	0.36	—	0.04	0.54	0.02
Skimmilk (dried)	7.6	1.29	0.97	—	—	1.46	—
Soyabean oil meal	5.6	0.26	0.73	0.28	—	2.20	0.07
Tankage (55–60% protein)	21.1	7.01	3.42	—	—	0.55	—
Tankage (50–55% protein)	27.1	11.70	5.15	—	—	—	—
Wheat	1.8	0.06	0.39	0.16	0.04	0.44	0.10
Wheat bran	5.8	0.12	1.26	0.58	0.09	1.23	0.07
Wheat middlings	3.9	0.10	0.88	0.40	0.14	1.12	0.04
Wheat shorts	3.7	—	—	—	—	—	—
Wheat feed flour	2.8	0.08	0.64	—	—	—	—
Whey (dried)	17.7	1.18	0.66	—	—	—	—

The important minerals in poultry nutrition, from a quantitative viewpoint, are calcium, phosphorus, chlorine, sodium, potassium and magnesium. In addition, iron, copper, manganese, iodine and probably others are required. Foods of plant origin are low in chlorine and sodium and, with the exception of the legumes, are low in calcium. Since the rations of poultry contain a fairly high percentage of cereal grains and their byproducts which are fairly high in phosphorus, there is little or no danger of aphosphorosis in poultry. Calcium is the mineral most likely to be deficient in poultry rations. It is possible that the "availability" of the phosphorus in cereals, one-half of which is phytin, may need some consideration. Phytin is the calcium-magnesium salt of inositol phosphoric acid and presumably the digestive juices of the fowl contain no enzyme capable of hydrolyzing the compound. Some feedstuffs contain an enzyme which can digest the substance. However, the fact that practical experience over a period of years has shown no indication of phosphorus being a limiting factor in poultry production, would suggest that either phytin is available to poultry or else the present accepted levels of phosphorus in the rations are so in excess of the requirement that the "non-

availability" of this salt is overcome. Feeds of animal origin are rich in all of the more important minerals required by birds and compensate for the deficiencies of foods of plant origin. The beneficial results attendant upon the inclusion of protein supplements of animal origin in poultry rations is due not only to the amount and quality of the protein which they furnish but to their mineral and, in some instances, to their vitamin content, as well. Concentrates such as meat meal and fish meal add appreciable quantities of minerals, particularly calcium and phosphorus, to rations. This fact is often disregarded and disastrous amounts of "mineral" supplements, such as bone meal or limestone, are added to the ration. The resultant excess amount of these minerals is decidedly detrimental.

It would appear that, in practical rations, only five minerals need consideration, namely calcium, phosphorus, sodium, chlorine and iodine. Halnan (129), from comparative analyses, has calculated that the body of the hen contains sufficient calcium for the formation of 10.7 two-ounce eggs, magnesium for 37.0, potassium for 62.6, sodium for 23.3, phosphorus for 107.0, sulphur for 34.2, chlorine for 29.0 and iron for 162.0. Jull (197) calculated that a hen fed on wheat alone would have to eat 12 pounds of wheat to supply the necessary calcium required for one egg, $\frac{1}{2}$ pound to supply the necessary sodium and $3\frac{1}{2}$ ounces to supply the chlorine. Lippincott and Card (236) calculated that a flock of 400 hens weighing 4 pounds each and in 50% production would require 404 pounds of corn daily to supply the calcium requirement for maintenance alone, whereas the eggs would necessitate over 6 tons of corn daily. These figures, although reducing the matter to an absurdity, nevertheless emphasize the necessity of supplementing cereal grains with minerals from some source, as well as pointing out the limitations of body reserves.

It was rather startling to the reviewer to discover that little or no work has been done with reference to the optimum total mineral content of poultry rations. Admittedly, the total mineral content of a ration, taken by itself, may be very misleading, since the results would depend upon the kind and amount of the individual minerals therein. However, it would seem possible by the use of certain basal diets together with the use of a "complete" mineral mixture to arrive at some base line. From a survey of results with simplified diets both in our own and other laboratories, and by a consideration of the voluminous literature dealing with perosis, the optimum level for chicks would seem to lie between 4 and 6%. A total mineral content in excess of 6% usually results in bone disorders. Parkhurst and McMurray (305) encountered perosis on a diet analyzing only 3.8% ash, but it only contained 0.19% calcium. The mineral requirements of laying hens are largely met in practise by allowing them unlimited access to oyster shell and bone meal.

3. CALCIUM AND PHOSPHORUS REQUIREMENTS

(a) General

In addition to the functions of calcium and its interrelationship with other minerals, which have been pointed out, it is necessary for the coagulation of blood and the clotting of milk during digestion. Phosphorus, in addition to its function in bone and tissue formation, and in the regulation

of the pH of the body fluids, is of vital importance in metabolism. Phosphorization of carbohydrates and fats is the primary step in their intermediate metabolism. Buckner and Harms (32a) investigated the effect of calcium supplements on digestibility.

(b) Calcium and Phosphorus for Growth and Bone Formation

The ratio between the amount of calcium and that of phosphorus in the ration would seem to be of some importance, since an excess of one results in depletion of the other for excretory purposes. An optimum Ca : P ratio in the ration apparently lessens the requirement for vitamin D, but with a vitamin D deficiency no Ca : P ratio will produce good bone. Moreover, some investigators seem to have lost sight of the fact that the absolute amounts of calcium and phosphorus often are more important, the ratio occurring as a natural sequence. It is quite possible to devise a ration with a good Ca : P ratio, but furnishing inadequate or excessive amounts of both minerals.

Bethke, Kennard, Kick and Zinzalian (21) concluded that a Ca : P ratio of 3–4 : 1 was optimum for calcification. A close inspection of their data indicates that the level of calcium in the ration had more effect than the ratio. Although Hart, Scott, Kline and Halpin (142) are often quoted as advocating a wide ratio, they concluded that, in the presence of adequate vitamin D, with the calcium varying from 0.61 to 1.13% and with 0.30% phosphorus, the optimum ratio was 2–4 : 1. With minimum vitamin D the best calcification was obtained with 2.49 to 2.71% calcium and 0.66 to 0.83% phosphorus, giving a ratio of about 3–4 : 1. Tully, Hauge, Carrick and Roberts (380, 381), Massengale (254), Marcq and Dervyst (250), and Holmes and Pigott (169) have dealt with this problem. Bethke, Kennard and Kick (20) obtained their best results with 0.86% Ca and 0.44% P, with a ratio of about 2 : 1. Wilgus (404) concluded that the Ca : P ratio may vary between 1–2 : 1; 2.5 : 1 was borderline; and 3.3 : 1 was disastrous. This problem has also been studied by Parkhurst and McMurray (304). In discussing this question, Titus, McNally and Hilberg (374) concluded that there was no common optimum ratio for all rations and that it depended partially on the acid-base balance of the diet. Mussehl and Ackerson (287) also concluded that the Ca : P ratio was not the only factor involved in bone formation and growth. It would seem feasible to conclude that with adequate vitamin D and reasonable levels of calcium and phosphorus in the ration, the Ca : P ratio may vary between 1–3 : 1 with 2 : 1 optimal.

The minimum calcium requirement for young chicks has been set by Wilgus (404) as 0.66%, although practical mashes generally contain about 1.2%; by Bethke, Kennard, Kick and Zinzalian (21) at 0.96–1.00% or or less; between 0.66 and 0.86% by Bethke, Kennard and Kick (20); at about 0.71 to 0.75% by Hart, Scott, Kline and Halpin (142); less than 0.94% by Supplee (367); 1.07 to 2.14% to eight weeks of age and 0.93 to 1.36% to twelve weeks by Sherwood (347) and 1.0 to 1.8% by Carver, Brunstad, St. John, Frasier and Athow (58). From analytical data, Mitchell, Hamilton and Card (277, 278) devised a calcium feeding standard for White Leghorn chicks varying from 0.11 grams per day at one-half

pound body weight to 0.14 grams at 5 pounds for cockerels. For pullets they recommend 0.10 grams per day at one-half pound body weight increasing to 0.19 grams at two pounds and then decreasing to 0.08 grams at 5 pounds body weight. The calcium requirements for growing turkeys range from 0.70 to 0.84% according to Mussehl and Ackerson (288).

The minimum phosphorus requirements have been reported as 0.50% or less by Wilgus (404), between 0.37 and 0.60% by Bethke, Kennard, Kick, and Zinzalian (21); Hart, Scott, Kline and Halpin (142) indicated that 0.30 to 0.42% was borderline; between 0.26 and 0.50% by Supplee (367); by Watkins and Mitchell (394) as less than 0.50%; while Sherwood (347) recommends 0.65 to 0.87% for chicks up to eight weeks and 0.48 to 0.53% to twelve weeks.

The calcium and phosphorus requirements have been studied also by Jull (195), Kupsch (219a), Gutowska (122a), and Kaupp (201).

TABLE 5.—CALCIUM AND PHOSPHORUS REQUIREMENTS FOR CHICKS OF LIGHT BREEDS*

Body weight lbs.	Calcium requirements			Phosphorus requirements		
	Net requirement grams per day	Dietary calcium required grams per day	Necessary calcium in dry ration, per cent	Net requirements grams per day	Dietary phosphorus required grams per day	Necessary phosphorus in dry ration per cent
Pullets						
0.5	0.09	0.18	0.64	0.08	—	0.32
1.0	0.13	0.26	0.70	0.12	—	0.35
1.5	0.15	0.30	0.68	0.15	—	0.34
2.0	0.15	0.30	0.59	0.16	—	0.31
3.0	0.12	0.24	0.39	0.15	—	0.25
4.0	0.05	0.10	0.14	0.07	—	0.07
Cockerels						
0.5	0.10	0.20	0.71	0.09	—	0.36
1.0	0.16	0.32	0.86	0.14	—	0.43
1.5	0.19	0.38	0.86	0.18	—	0.43
2.0	0.20	0.40	0.78	0.20	—	0.39
3.0	0.20	0.40	0.66	0.21	—	0.34
4.0	0.15	0.30	0.42	0.20	—	0.28
5.0	0.09	0.18	0.22	0.13	—	0.16

* Data of Mitchell and McLure.

Ackerson, Blish and Mussehl (2), using a ration containing 0.70% calcium and 0.81% phosphorus found that 43.4% of the calcium and 27.3% of the phosphorus intake was retained. Later, these workers (3) reported that utilization of phosphorus, the ration containing about 0.80% was unaffected by varying the calcium content of the diet, the retention being about 28%. The percentage of calcium retained was 35% when the diet contained 0.9%, 24% when the level of dietary calcium was 1.5%, and 13% when 2.3% was supplied.

From a resume of this evidence the minimum calcium requirement for growing chicks would seem to be about 0.70%, and that of phosphorus

as 0.40%. To provide a margin of safety, without danger of overdosage, practical chick rations probably should furnish about 1.0% calcium and 0.50% phosphorus. Mitchell and McClure (280) have estimated the requirements for growing Leghorn chicks as shown in Table 5. Larger breeds probably have somewhat greater requirements than this light breed. However, since their needs are expressed in percentage of dry matter in the ration, and larger breeds consume more feed, these percentage estimates would probably suffice.

(c) Calcium and Phosphorus for Laying Hens

The calcium requirements of the laying hen will depend on the number of eggs laid, the amount of calcium in the egg exceeding that of other minerals. An average egg contains approximately 2 grams of calcium, the greater part being present in the shell, only 0.026 gram being present in the yolk and 0.004 gram in the white. Compared with bone calcification, egg shell formation is an extremely rapid process. The calcium carbonate is secreted by the glands in the wall of the oviduct and according to Cruickshank (82) the process requires only 12 to 16 hours. The calcium is supplied by the circulating blood, since the oviduct itself has little or no reserve of calcium.

In the absence of a sufficient supply of calcium, egg production is lowered and may finally cease, the egg shells becoming progressively thinner. The serum calcium of the hen falls and there is a depletion of the calcium and phosphorus of the bones and a reduction in hatchability. Collier (69), Wheeler (399), Buckner and Martin (33), Buckner, Martin and Peter (42, 44, 45, 47, 50) and Buckner, Martin, and Insko (39) and Buckner, Martin, Pierce and Peter (51) have studied this problem. The percentage composition of the shell, however, remains constant showing that, like bone formation, if the mineral is deposited, it is laid down in the normal proportion. Deobald, Lease, Hart and Halpin (87) concluded that about 10% of the calcium stored in the bones may be called upon for egg shell formation. The phosphorus requirements are not greatly raised for egg production.

The calcium and phosphorus requirements of laying hens have not been the subject of as many studies as those of chicks. Norris, Heuser, Wilgus and Ringrose (293) concluded that 1.65% of calcium is just sufficient to meet the requirements as judged by egg production, shell strength and ash, and by the levels of calcium and phosphorus in the blood. On a ration containing about 1.8% calcium, 0.5% phosphorus was insufficient, but 0.75% was adequate. Miller and Bearse (271) using rations varying in calcium from 2.23 to 3.03%, and in phosphorus from 0.40 to 1.08%, concluded that egg production was influenced by the amount of phosphorus in the ration. They obtained the highest egg production with 0.80%. From their data, it would appear that 2.25% calcium was adequate or more than adequate. Mitchell and McClure (280) have prepared a standard for calcium and phosphorus feeding for laying hens, part of which is given in Table 6. Although it varies according to the rate of egg production it is based unfortunately upon a three-pound hen, a weight generally below that encountered in the field. Until more data

TABLE 6.—CALCIUM AND PHOSPHORUS REQUIREMENTS OF LAYING HENS*‡

Rate of egg production	Net calcium required, grams	Necessary calcium† in dry ration, per cent	Net phosphorus, grams	Necessary phosphorus† in dry ration, per cent
100	2.10	4.16	0.27	0.38
75	1.60	3.52	0.24	0.35
50	1.11	2.74	0.21	0.32
25	0.61	1.72	0.18	0.30
0	0.12	0.39	0.15	0.25

* Data of Mitchell and McClure.
† Based on 50% utilization.
‡ White Leghorn hen weighing three pounds.

are available, the safe practise would seem to be that of allowing the birds free access to such supplements as oyster shell or limestone and bone meal.

Titus, Byerly, Ellis and Nestler (372) in a series of experiments also investigated this requirement by varying the levels of calcium and phosphorus in diets fed to laying hens and pullets. In one series the phosphorus level was constant at 1.2% and the calcium was fed at 1.2, 3.0 and 5.4% levels; in another the phosphorus was maintained at 0.9% with the calcium at 0.9, 2.25 and 4.05%; in a third the phosphorus was constant at 1.2% and the calcium intake was 1.3, 3.0 and 5.4%. Their results establish the fact that the calcium requirement of pullets and hens is different and that a high level of calcium adversely affects the hatchability. In disagreement with the Washington results they concluded that the level of phosphorus intake had no effect on egg production. The "proper" quantity of calcium in the diet varied from 1.95% for crossbred hens to 2.20% for Leghorn pullets, when the phosphorus content was 0.9%.

The Beltsville workers have devised a formula for estimating the calcium requirement of laying birds. The equation is:

$$\mathrm{Ca} = \frac{1.292(\mathrm{P}F - 10.8E) + 200E.}{F}$$

in which

Ca = the required percentage of calcium in the diet,
1.292 = the ratio of Ca to P in dicalcium phosphate,
P = the percentage of phosphorus in the diet,
F = the number of grams of feed consumed by a laying chicken during a year,
10.8 = 100 times the quantity (in grams) of phosphorus in the egg,
E = the number of eggs produced in one year,
200 = 100 times the quantity (in grams) of calcium in an average egg.

In the reviewer's opinion, the practice of mixing mineral supplements into laying mashes is to be condemned, since it does not make full allowance for variation in the rate of production, nor of size of egg. The method of controlling calcium intake, as outlined above, may be theoretically sound, but its application in practise is not feasible. Moreover, it is obvious

that such a method cannot be used by feed manufacturers, and the reviewer is doubtful if sufficient data on feed consumption, etc., is available to enable even the research worker to use the formula. After all, any such formula is based upon the flock average, and the high producers will be underfed, the low producers will be overfed—surely an unscientific procedure.

(d) Calcium and Phosphorus Metabolism During Production

(1) Calcium

With the onset of egg production the calcium content of the blood of the female may rise to double the normal concentration maintaining a high though variable average during the laying period, falling during moulting. In the male no such changes occur, the calcium level remaining constant at about 10 to 11 mg. per 100 cc. serum. Bell (10) found the calcium index in the case of a non-laying hen to vary between 0.9 and 1.3, whereas in the laying hen the variation was from 0.5 to 3.1. Buckner, Martin and Peter (46) and Buckner (32) showed that there was not enough calcium in the shell gland to account for all the calcium in the shell, and postulated a direct removal from the blood during shell formation. Hughes, Titus and Smits (179) showed that the level of calcium was much higher in laying hens—up to three times—than in immature pullets. Parhon (302) found an increase in serum calcium during laying which diminished with the onset of broodiness. Russell, Howard and Hess (330) reported that the serum calcium of hens lay between 13.0 and 26.7 mg. Ca per 100 cc. serum if an ovum greater than 1 cm. in diameter was present. When no ova were observed the level was between 7.5 and 13.0 mg. MacOwan (266) found that with a 1 gm. ovum the serum Ca was 13.4 mg., with ova weighing up to 30 gm. it was 23.5, while beyond this size of ovum the level was about 20.7 mg. per 100 cc.

Buckner, Martin and Hull (36) showed that the laying hen absorbed calcium at a rapid rate from the intestine, the calcium content of the blood in the anterior mesenteric vein being 2 to 2.5 mg. per 100 cc. higher than in the anterior mesenteric artery. This difference was not found in non-laying hens.

Charles and Hogben (61) could not correlate the level of serum calcium in the blood with the position of the egg in the oviduct. They found wide variations in the blood calcium when the egg was present in the oviduct, showing a range of 10.5 to 28.5 mg. However, they used a different hen for each determination, and the level of blood calcium varies greatly among individual hens. With no egg in the oviduct there was less variation, from 14.7 to 19.6 mg. Knowles, Hart and Halpin (215) reported a variation in the calcium in the blood of laying hens from one oviposition to another, using the same hen for each series of bleedings, but the maxima and minima were not constant for the same hen, the period of low level corresponding to the time for shell deposition, and the high level corresponding to the absence of a fully-formed egg in the shell gland. They also found, using one hen, a marked difference in the calcium level in the blood from the uterine artery and vein during shell deposition. However, Deobald, Lease, Hart and Halpin (87) repudiated these previous findings and stated that the calcium content in the blood of the individual hen was

nearly constant, varying only within narrow limits during a complete egg cycle, a finding substantiated by Feinberg, Hughes and Scott (106). Rochlina (328) has also studied this problem. In a recent study Deobald, Christiansen, Hart and Halpin (84) removed various parts and the entire oviduct from pullets. The blood calcium curve at maturity was normal, showing that the oviduct itself had no influence on the mobilization of calcium previous to laying.

Benjamin and Hess (11), Correll and Hughes (77), Taylor and Russell (370), and Laskowski (228, 229) have shown that the rise in total calcium is not associated with the diffusible calcium of the blood, which remains constant in laying and non-laying hens. The adsorbable diffusible fraction decreases slightly during egg production and the non-adsorbable fraction increases slightly, so that the level of total diffusible Ca remains the same. Both fractions of the non-diffusible Ca increase greatly, according to Benjamin and Hess (11), Paul (306), and Heller, Paul and Thompson (153). Greenberg, Larson, Pearson and Burmester (120) also concluded that the very large increase in the serum calcium level was confined almost exclusively to the non-diffusible fraction. The increment could only be partly accounted for by the formation of a colloidal form of calcium phosphate.

Calcium metabolism of the egg during incubation has been studied by Plimmer and Lowndes (318), Buckner, Martin and Peter (45), Gazzaniga (116), Insko and Lyons (188), Glaser and Piehler (117a), and by Kamachi (198).

(2) Phosphorus

As shown by Heller, Paul and Thompson (153), during the first five months the total P content of blood rises gradually from 110 mg. to about 115 mg. per 100 cc. With the onset of egg production there is a sudden increase to a level of 131 mg. which persists throughout production. Similar changes take place in the total P of cells and plasma, because of an increase in the lipoid fraction in both. Greenberg, Larson, Pearson and Burmester (120) verified the striking increase in the fat and phospholipid content of the blood during ovulation, as reported by Laurence and Riddle (231) and Warner and Edmund (393). Roepke and Hughes (329) and Laskowski (230) reported that while in non-laying hens and males the total P is equal to the sum of the lipoid and acid-soluble fractions, in the laying hen the total phosphorus is greater than the sum of the two, owing to the appearance of a new fraction, a phosphoprotein, having properties similar to vitellin. Presumably, the increase in lipoid phosphorus and the appearance of phosphoprotein is associated with the formation of yolk, which contains considerable lecithin and vitellin.

According to Laskowski (230) the inorganic blood phosphorus also increases during the laying period, although there is considerable individual variation. Roepke and Hughes (329) found a higher value for the inorganic P in the blood of laying as compared with non-laying hens, but it was somewhat below the value for males. Greenberg, Larson, Pearson and Burmester (120) reported an increase in inorganic serum phosphorus, which was directly correlated with the serum calcium level. Deobald, Christiansen, Hart and Halpin (84) reported that the total phosphorus in

the blood showed a direct correlation with the blood calcium curve, except at the high peak immediately preceding the laying period. Feinberg, Hughes and Scott (106) reported a marked rise during the period of shell deposition. Heller, Paul and Thompson (153) and Benjamin and Hess (11) claimed that the inorganic phosphorus was similar in cockerels and laying hens.

Benjamin and Hess (11) reported that the filtrability of the inorganic P was very low and is only very slightly reduced in hypercalcaemia, whereas Laskowski (230) found that inorganic P in the plasma of immature pullets was almost entirely diffusible, but that this diffusibility was decreased one half in laying hens. Similar results were obtained by Roepke and Hughes (329), Grollman (121) and Greenberg, Larson, Pearson and Burmester (120). Their values are shown in Table. 7.

The phosphorus metabolism of the developing embryo has been studied by Plimmer and Scott (317), Baldwin and Needham (9), Insko and Lyons (188), Kamachi (198) and by Schmidt (340).

(e) Phosphatase

Phosphatase is an enzyme which effects the hydrolysis of certain phosphoric esters, magnesium acting as a co-enzyme, present in blood, bone, intestines and kidney. This enzyme is considered to be an active agent in ossification, bringing about the hydrolysis of these esters in the blood and thereby raising the concentration of inorganic phosphate so that calcium phosphate is deposited. Emslie (101) Schecter (334), and Common (73, 75) have found that the plasma phosphatase of chicks suffering from experimentally producted "rickets" is increased above normal. Confirmatory evidence has been obtained in the reviewer's laboratory. This

TABLE 7.—PARTITION OF THE CALCIUM AND INORGANIC PHOSPHORUS IN THE BLOOD SERUM OF THE FOWL*

		Calcium			Inorganic phosphorus		
	Age	Total	Diffusible	Non-diffusible	Total	Diffusible	Non-diffusible
Non-laying	½ month	11.3	6.4	4.9	10.0	8.5	1.5
	1 month	12.0	7.0	5.0	8.8	7.4	1.4
	2 months	12.9	7.0	5.9	8.7	5.8	2.9
	3 months	12.0	6.9	5.1	6.9	5.4	1.5
	4 months	12.1	6.8	5.3	6.4	5.0	1.4
	5½ months	11.5	9.6	1.9	5.5	5.6	0 0
Pre-laying	6–7½ months	31.6	8.8	22.8	7.4	3.6	3.8
Laying	6½–8 months	23.7	9.4	14.3	6.1	3.9	2.2

* Data of Greenberg, Larsen, Pearson and Burmester.

finding is important since Branion (30) has shown that the plasma phosphatase of rachitic rats is generally decreased. Since it is well established that "rickets" can be induced in chicks by deprivation of vitamin D alone, whereas to produce the disease in rats it is necessary to deprive them not

only of the vitamin but also to upset the Ca : P ratio of the diet, it is not surprising that the two conditions are fundamentally dissimilar. Moreover, Kay (203) has shown that in bone disease of humans the plasma phosphatase is increased.

Common (75) found that the serum phosphatase while showing considerable individual variation, was significantly higher in laying than in non-laying hens or cocks. Auchinachie and Emslie (8) did not observe any difference in the values for "strictly normal" laying and non-laying hens but with birds confined indoors with inadequate vitamin D, laying pullets showed a much higher plasma phosphatase value. This would support the contention that, under certain conditions at least, the laying hen draws on her skeleton for calcium, with resultant osteoporosis (Doyle, 89). Hall and King (125) found that the bone phosphatase content of rachitic chicks was about twice the normal value whereas in hypervitaminosis D it was reduced to one-sixth normal.

(f) Parathyroid Glands

Since the parathyroid glands, two small bodies at the posterior pole of each thyroid gland, produce a hormone concerned in the regulation of blood calcium, it does not seem amiss to consider them in this review.

Doyle (89) and Hogan, Shrewsbury and Kempster (166) noted an enlargement of the parathyroid, up to 5 to 8 times the normal size in the absence of vitamin D. According to Higgins and Sheard (162) and Sheard and Higgins (345), both hyperplasia and hypertrophy exist, the former being more marked. In severe cases, the parenchyma shows regression and cysts develop, according to Higgins, Sheard and Wilder (163) and Nonidez and Goodale (292). When the rachitic birds are irradiated the parathyroids return to normal size.

Collip (70) was unable to demonstrate any effects from the injection of parathormone upon non-laying hens, but Knowles, Hart and Halpin (215) were able to show that chickens respond to a single, large injection (30–60 units) of the hormone, but in order to ascertain the rise the blood samples must be taken early after injection. They concluded that the rise in blood calcium as a pullet comes into production is due primarily to increased activity of the parathyroid. Deobald, Lease, Hart and Halpin (87) corroborated this finding but observed that long continued injections of the hormone into hens starved of calcium was without effect. Deobald, Christiansen, Hart and Halpin (84) reaffirmed this conclusion, and further reported that hens on a low calcium ration did not respond even when calcium carbonate was administered previously. Low calcium and low phosphorus diets resulted in similar blood calcium curves. Both rations inhibited egg production and both caused a negative reaction to parathormone. Altman (6) obtained a rise in serum calcium in non-laying birds by injections of parathyroid extract, progynon B, egg yolk, liver extract and to a still higher level by simultaneous injection of anterior pituitary extract and egg yolk. Injection of the follicle-stimulating fraction of the anterior pituitary with parathyroid extract gave no greater rise in serum Ca than parathyroid alone. Egg yolk raised the blood calcium of capons. The blood calcium was depressed by injections of the thyrotropic hormone of the anterior pituitary and by simultaneous injections of extracts

of the whole anterior pituitary and parathyroid, while anterior pituitary extract alone was without any effect. The elevated blood calcium level of laying hens was decreased by injections of progynon B and by egg yolk.

By histological investigation, Sun and MacOwan (366) found that the gland in laying hens differed from that of the non-laying hen. In the former, the cells were larger, less closely packed, with some degree of vacuolation. Later MacOwan (266) confirmed this and was able, by injections of parathormone, to increase the serum calcium in pullets, but obtained a varied response with non-laying hens and no response in males.

(g) Mineral Balance Experiments

Although mineral "balance" or retention experiments with fowl have yielded some valuable results in calcium and phosphorus metabolism, only very few such studies have been carried out. The technical difficulties involved are great. Such studies are complicated by the fact that these minerals are excreted into the intestine, that the urine and faeces are voided together, and by the fact that calcium may remain for sometime in the gizzard in the form of oyster shell or certain grits—to say nothing of the practical difficulties in preventing food spillage and egg breakage. Moreover, even with hens in steady production, it normally takes from 21 to 27 hours for the egg to pass through the oviduct, and it may be "held" for some hours longer. The question arises as to which day the calcium and phosphorus in an egg laid should be credited. A "short cycle" as against a "long cycle" hen further complicates the problem. Again, most of the phosphorus is in the yolk, and the time necessary for the "maturing" of the yolk is also involved. It should be evident that one should not be too critical of any discrepancies in results of such studies which arise. In fact, it is astonishing that such good agreement has resulted.

The hen has no large reserve of calcium or phosphorus for egg formation, and the storage of these two elements occurs about one week prior to the onset of laying according to Halnan (127), or about two weeks according to Common (71). This increased demand is met by increased retention from the food, the percentage retention of calcium rising from 20 to 70%, provided there is an adequate dietary supply of both calcium and salt. According to Stewart (360), Osawa (301), and Mayneord (261) the calcium in egg shell is mainly present as crystals of calcite. Halnan (129) offers an ingenious explanation for the mobilization of calcium for egg shell formation and its subsequent deposition. He postulates that the immediate precursor of the calcium carbonate of egg shell is calcium bicarbonate. The calcium absorbed from the gut increases the blood calcium content, which is maintained partly by the hormone of the parathyroids, acting by withdrawal of calcium from the bones and partly by the antirachitic vitamin by protecting the blood calcium from excretion. The larger part of this calcium is converted to a non-diffusible calcium-phosphorus complex which can be conveniently transported without upsetting the normal ionic concentration of the blood serum. The calcium secreted by the shell gland from the diffusible calcium of the blood serum reduces the ionic concentration of the serum calcium, thus favouring the break up of the non-diffusible calcium-phosphorus-protein complex. The diffusible

calcium concentration of the serum is restored to normal and the excess diffusible phosphate is excreted. Partial support for this theory is available from the finding of Halnan (127), Russell and McDonald (332), and Common (71) that there is greatly increased excretion of phosphorus during egg production, which the last worker correlated with the period of shell formation. Halnan (127) thought the phosphorus excretion during egg production was due to the formation of egg fat from phosphatids. Believing that the amount of calcium which could be held in reserve in the blood was small, Common (71) suggested that the skeleton acts as a temporary storehouse for this element. Withdrawal of CaO from the bones would necessitate the removal of equivalent amounts of P_2O_5 as a result of the breakdown of $Ca_3(PO_4)_2$. His later studies (72, 74) indicated that the voiding of phosphorus is dependent upon the amount of calcium in the diet, since in the presence of an adequate supply, heavy phosphorus excretion may not accompany egg production. It would seem that removal of calcium from the bones is not necessary for a hen adequately supplied with calcium carbonate and vitamin D. However, it is probable that this process does go on to some extent, particularly in the case of the heavy producer, or at the peak of production. Under such circumstances, it is doubtful if the hen would be able to absorb and assimilate calcium from the gut fast enough to meet the heavy requirement for shell formation. During the moult or in periods of low production the skeletal reserve would be restored. We have found that laying birds, although having unlimited access to oyster shell, consume appreciable amounts of bone meal in addition, suggesting that there is some physiological requirement for phosphorus during egg production. The bones of a laying bird, ingesting suboptimal amounts of calcium or vitamin D presumably are osteoporotic. Schmidt (339) has also investigated calcium and phosphorus retention. This problem has also been discussed by MacDonald and Orr (239).

With regard to the combination in which calcium and phosphorus occur in the excreta, Halnan (127) considered that during non-laying, the actual values for excreta calcium agreed with that theoretically required to combine as tricalcium phosphate with all the phosphorus in the droppings. During the laying periods the phosphorus excretion was in excess of that required for such combination. Russell and McDonald (332) found that during production there was more phosphorus excreted than is necessary to form tricalcium phosphate, and during non-production less than required to form this compound with the excretory calcium. Knowles, Watkin and Hendry (216), using non-layers, concluded that the phosphorus was present as dicalcium phosphate, the remaining calcium being present as the carbonate. Common's (72) experiments show that, during egg laying, and in only a few instances during non-production, all the phosphorus could not have been present as dicalcium phosphate, and generally, there was insufficient calcium to combine with all the calcium as the monocalcium salt. He concluded that the extra phosphorus was excreted in the urine, probably as a soluble ammonium salt. Morgan (281) concluded that pullets, fed on the usual laying ration, receive sufficient phosphorus to meet their requirements as indicated by the cumulative positive balance. During heavy egg production, most of the birds were in negative calcium balance, particularly during the first months of production.

(h) "Availability" of Various Ca and P Compounds

As was pointed out, the term "availability" means very little; nevertheless it is used here in the practical sense to indicate whether various calcium and phosphorus compounds will serve as a source of these elements for bone formation or egg production. Bethke, Kennard and Kick (20), using rations containing 0.86% calcium, found no difference in the availability of the calcium in the carbonate, sulphate, lactate and both di- and tricalcium phosphate salts or the commonly used minerals of limestone, steamed bone meal, rock phosphate, phosphatic limestone, or oyster shells for bone formation. As will be pointed out later, rock phosphate and phosphatic limestone, often give unsatisfactory results due to their fluorine content. Similarly some limestones, particularly dolomite, are inferior due to their magnesium content. Phosphatic limestones contain varying proportions of calcium phosphate and carbonate, but "rock phosphate" usually is applied to material containing from 60 to 75% calcium phosphate whereas "phosphatic" limestone contains about one-third as much phosphorus. Lewis (234) observed that bone meal was superior to inorganic phosphate. Buckner and Martin (35) reported that tricalcium phosphate, fed alone or with calcium sulphate or carbonate gave increased growth and bone development, although it did not materially influence the percentage of ash, calcium and phosphorus in the bone. Calcium carbonate, calcium sulphate and tricalcium phosphate served equally well as sources of calcium. Later, Buckner, Martin and Peter (42) showed that the calcium in rock phosphate could be utilized for bone formation. In other publications, (Buckner, Martin and Insko, 38) they confirmed these results using limestone, raw bone meal, rock phosphate and dicalcium phosphate. Deobald, Elvehjem, Hart and Halpin (86) also found that the bone ash content was higher when the supplement contained phosphorus in conjunction with calcium. Although growth differences occurred, the calcium in limestone, gypsum, rock phosphate, calcium gluconate, "dicapho", steamed bone meal, and dolomite was equally available in spite of differences in solubilities of these supplements. When the vitamin D intake was borderline, the soluble salt calcium lactate, gave better growth and bone formation than the insoluble calcium silicate, whereas with adequate D no difference was apparent. When extremely insoluble calcium silicates were fed their availability was proportional to their solubility in dilute acids, increased amounts of the antirachitic vitamin having no influence. Using a rachitogenic diet, Higgins and Sheard (162) concluded that better growth and development took place in chicks fed tricalcium phosphate than in those fed dicalcium phosphate.

Buckner, Martin and Peter (49) concluded that limestone or oyster shell serve equally as well as sources of calcium for laying hens but the calcium in rock phosphate (tricalcium phosphate) could be utilized for growth of bone but not for egg shell formation. Fluorine was probably a factor in this result. Hunter, Dutcher and Knandel (181) found that calcium carbonate and calcium gluconate function with equal efficiency as sources of calcium for the growing chick, but for the laying hen the calcium gluconate was slightly more efficient. Kistler (211) observed that bone was superior to acid phosphate or raw rock phosphate in the production of eggs.

Buckner, Martin and Peter (49) compared calcium lactate, chloride, sulphate, and tricalcium phosphate with calcium carbonate, concluding that the calcium carbonate is more effective in egg production as judged by the weight of the egg contents and shell. Calcium chloride was not well tolerated. On the other hand, Fangauf and Kallman (105) obtained good results with this salt. Russell and McDonald (332) found that the laying bird can utilize the calcium from calcium citrate as well as from the carbonate for egg formation. Massengale and Platt (256) reported that birds receiving c.p. calcium carbonate as a source of calcium did not grow as well or lay as many eggs and produced eggs with less shell than those receiving oyster shell or limestone. Calcium carbonate in the form of oyster shell or limestone was better utilized than tricalcium phosphate, but the latter was superior to c.p. calcium carbonate. Limestone was somewhat superior to oyster shell. Buckner, Insko and Martin (41) concluded that marls served as satisfactory sources of calcium for laying hens.

Waite (391) found no difference in the utilization of the calcium from limestone and oyster shell for egg shell formation. Dougherty and Grossman (88) compared limestone with oyster shell and limestone combined, and found that the production was superior in the group receiving limestone alone, although the egg shells were equally good in both groups. Oyster shell was found by Kennard (205) to be superior to limestone, while Hart, Steenbock and Morrison (144, 145) recorded better egg production with oyster shell than with limestone, clam shell, dry bone, or quartz. Iowa workers (190) reported, from a comparison of oyster shell, clam shell and limestone, that the clam shell was inferior to the other two; similar results were obtained by Hart and Halpin (141), whereas Fangauf and Bruninghaus (104) could find no advantage in replacing cheap mussel shell with oyster shell.

Alder (4) compared oyster shell, blue limestone, dolomite, oolite and calcite, and found dolomite decidedly inferior because of its high magnesium content, with little or no difference between the other minerals. Tully and Franke (378) found no significant difference as judged by egg production, egg weight, and shell breaking strength between oyster shell, Black Hills limestone, dolomitic limestone, Meno chalkstone, clam shell, commercial limestone and commercial calcite. Martin and Insko (252) concluded that white shiny limestone was more attractive to birds than dull grey limestone, yet both were adequate sources of calcium for egg shell formation. Halpin and Lamb (133) reported that high levels of raw rock phosphate and phosphatic limestone were detrimental. Miller and Bearse (272) reported no appreciable differences in egg production, shell quality, egg weight, mortality, body weight and hatchability between lots of birds fed eastern oyster shell (Atlantic), clam shell, western oyster shell (Pacific), western limestone (Washington) and aragonite as sources of calcium. Egg production was significantly lower in the lot fed phosphatic limestone but in other respects it was the equal of the other lots. Since this supplement contained 1.3% magnesium, it is debatable whether its detrimental effect was due to this element or to fluorine. In one lot fed dicalcium phosphate in place of bone meal, along with eastern oyster shell, the production was lower. Guisti (122), Kitto (211a), Voelcker (390a), and Edin and Anderson (95) have also studied this problem.

From a consideration of the above reports, it would seem evident that the various commonly used sources for calcium and phosphorus supplements can be considered to be of equal value both for bone formation and egg production, with the exception of rock phosphate and phosphatic limestones which may be contaminated with fluorine, and of dolomite limestones on account of the magnesium content.

(i) Anticalcifying Properties of Cereals

King and Hall (210) concluded that the addition of oat groats (oats with the hulls removed) induced a more severe rachitic condition in chicks. Prolonged boiling of the groats with hydrochloric acid, without subsequent dialysis, appeared to render the anticalcifying substance more available. Vitamin D tended to overcome this effect. Unpublished data from the reviewer's laboratory, of a study involving corn, wheat, oats, oat groats and barley with graded levels of irradiated ergosterol, failed to show this anticalcifying property of oats. No cereal could be singled out as possessing this property in excess of the others. Lachat and Palmer (225) repeated the experiments of Hall and King but were unable to show that oat groats was more rachitogenic than yellow corn. However, using the A.O.A.C. diet they showed that oats and corn are rachitogenic, the former appearing to be more rachitogenic when the rations are otherwise only mildly rickets producing.

4. PEROSIS

Perosis, also known as slipped tendon, deforming leg weakness and hock disease, is a leg abnormality of young growing chicks. Since there has been some confusion in regard to the symptoms, a brief description, taken chiefly from Milby (269) is given. The joint which is affected, commonly called the hock joint, is the tibiometatarsal joint. Some workers incorrectly refer to this as the tibia tarsal joint—strictly speaking, it is the tibio-tarso-metatarsal joint. Bradley (27) describes the bones concerned as follows: "The distal end (of the tibia) has two articular elevations separated by a smooth groove. Strictly speaking, this part of the tibia represents the proximal row of the tarsal bones and the whole bone should therefore be called the tibio-tarsus. In the adult there is no independent tarsus. In the embryo there are indications of two rows of elements, but before long the proximal row fuses with the tibia and the distal with the metatarsus. The adult metatarsus, or tarsometatarsus is represented by one long bone composed of second, third and fourth metatarsal bones in union." In the normal chick the tendons at this joint glide between two lateral condyles on the distal end of the tibia, which is perfectly straight. In chicks afflicted with perosis, the first observable symptom is usually a slight puffiness about the joint. At this time the tibia is usually straight but the metatarsus may or may not be slightly bowed. In many cases, between the ages of 2 to 10 weeks, generally between 4 and 6 weeks, the tendons, particularly the gastrocnemius or Achilles tendon, slip from the condyles to one side or the other, and one or both legs may be affected. In other cases, the hock becomes flattened laterally and the legs are bowed but the tendons do not slip. Frequently the articular cartilage at the distal end of the tibia appears to be slipped slightly from its normal position.

Often the chick is unable to stand but rests on its hocks with the afflicted leg extended to the front or side. The bones of the legs, particularly the tibia and metatarsus become deformed to a greater or less extent, dependent upon the severity of the condition. The epiphysis at the distal end of the tibia is bent laterally in the direction in which the tendon slips. Milby (269) concluded that this bending was the result, rather than the cause, of the displacement of the tendon. However, Titus (371) claimed that at twelve weeks of age, when union of the epiphyses and diaphyses of the tibiae of normal chicks begins, these are ununited in chicks with perosis, a conclusion contrary to that of Hall and King (124).

This condition was differentiated from rickets by Hogan, Shrewsbury and Kempster (165), although a condition described by Mitchell, Card and Carman (275) and by Mussehl, Blish and Ackerson (289) was probably this deformity. It has been clearly demonstrated by numerous workers that vitamin D has no relationship to perosis, but in the earlier work on rickets in chicks perosis may have been a complicating factor. Battery brooders tend to aggravate the condition, although they are not primarily responsible, and the heavier breeds of chicks seem to be more susceptible than the light breeds. The reviewer saw this abnormality among chicks from the same hatch in the same battery room and fed the same ration in which almost every Jersey Black Giant chick and about one-half of the Barred Plymouth Rock chicks were afflicted, but White Leghorn chicks showed little or no evidence of the condition. As will be pointed out, the blood and bone analyses of perotic chicks are normal. Excessive amounts of minerals, particularly phosphorus, are associated with the condition.

Payne, Hughes and Leinhardt (308) could find no correlation between the inorganic calcium and phosphorus in the blood nor in the ash, magnesium, calcium nor phosphorus content of bone and perosis. A histopathological study of the organs and tissues revealed no abnormalities, so they concluded that the condition was of an anatomical rather than a histological nature. Hall and King (124) found normal serum calcium, and inorganic phosphorus, bone ash and bone phosphatase values. Holmes, Pigott and Moore (174) were unable to reveal any significant difference between the size and mineral content of tibiae from slipped-tendon chicks and those of normal chicks. They concluded that slipped tendons were not primarily due to faulty mineral metabolism—it is presumed that by faulty mineral metabolism, the authors referred to abnormal calcification of the leg bones. Similar findings were reported by Wilcke (402), Milby (267, 269, 270), Heller and Penquite (156), Herner and Robinson (160), and also in the reviewer's laboratory.

Hunter and Funk (182) reported a high incidence of slipped tendon among chicks fed diets containing relatively large amounts of meat meal, meat and bone meal, or bone meal. Later, Hunter, Dutcher and Knandel (180) produced the abnormality by the addition to the ration of such materials as bone meal, sodium phosphate, calcium carbonate, and mineral mixtures. Decreased amounts of these minerals lessened the trouble. They made a very pertinent observation: "the presence of protein concentrates rich in inorganic salts, aggravates the condition when salts or salt mixtures are fed at the same time". Card (57) also warned against excessive mineral feeding. Payne, Hughes and Leinhardt (308) found that a

mineral mixture of c.p. calcium phosphate and calcium carbonate produced the same deleterious effects as equivalent amounts of bone meal and concluded that an important etiological factor was an excessive amount of either phosphorus or calcium or both. Titus (371) using a ration containing 1.42% calcium and 0.92% phosphorus (with a total ash content of 5.7%) was able to produce perosis in 42.5 to 85.0% of the chicks to which it was fed. Perosis-producing diets have also been developed by numerous workers including Sherwood and Couch (349), Heller and Penquite (154), and a "synthetic" diet by Clifcorn, Elvehjem and Hart (65).

Herner and Robinson (160), Hunter, Dutcher and Knandel (180), Schaible, Moore and Conolly (337), Parkhurst and McMurray (305), Roberts (327) and Solun and Arsenjaw (356) as well as others previously mentioned, concluded that the etiological factor involved in perosis was excessive mineral feeding. Milby (268) reported from a statistical analysis that the level of calcium in the ration was not significantly correlated with perosis but the amount of phosphorus was positively correlated. However, he cautiously pointed out that these findings "do not justify the conclusion that phosphorus is the chief causative factor of slipped tendon, but merely indicate that high percentages of slipped tendon are associated with high phosphorus in the ration. This warning was probably caused by his earlier finding (267) that, although the amount of phosphorus in the ration was highly correlated with the percentage of slipped tendon, the number of slipped tendons which were produced by various levels of inorganic phosphorus was much less than would be expected on the basis of the phosphorus content of the ration. Later, Milby (269) again stated "the incidence of slipped tendon increased as the phosphorus increased. Though the trend was very definite, the relation between the amount of phosphorus in the ration and the percentage of slipped tendon was not exactly linear, indicating that factors other than the amount of phosphorus in the ration may have been operating to influence the incidence of slipped tendon." Incidentally, he encountered perosis in 24% of chicks fed on a ration containing only 0.43% phosphorus. McGowan and Emslie (264) believed that the preventive action of certain feeds was due to their content of organic or "delayed action" type of phosphorus. Insko, Sowell and Lyons (189) reported that a widening of the Ca : P ratio, with its accompanying increase of calcium in the ration from 0.4 to 3.1 : 1 did not increase the percentage of slipped tendon. Increasing the phosphorus content of the ration, by the use of bone meal, raised the incidence of perosis, although the Ca : P ratio was approximately the same. They concluded that the amount of phosphorus fed might be one of the causative factors. Hammond (136) found a high correlation between perosis and total phosphorus in the ration, and a negative correlation between organic phosphorus or calcium and perosis. He concluded that inorganic phosphorus in the ration was the primary factor. Milne (274) also associated a high phosphorus level in the ration with a high incidence of perosis. However she pointed out that the form in which the phosphorus is present may be the determining factor, since the substitution of meat meal and fish meal for milk caused a marked increase in perosis. Unpublished results from the reviewer's laboratory indicate that the mineral in meat meal is more productive of perosis than a similar amount furnished by fish meal. Maw (260) also concluded that bone char has some property which causes a reduction of slipped tendons

when compared with a similar amount of bone meal in the ration. Similarly when meat meal was replaced by fish meal the number of slipped tendons was reduced. Wilgus, Norris and Heuser (407) reported that the severity of perosis was as great when the calcium and phosphorus content of the diet was increased to 1.5% and 1.0% respectively by the use of steamed bone meal, as when higher levels were fed. C. p. calcium carbonate, hydroxide and chloride as well as c.p. mono-, di- and tricalcium phosphate increased the severity to about the same extent as bone meal, although in their earlier paper (405) they reported that c. p. monosodium phosphate produced more severe perosis than steamed bone meal or technical tricalcium phosphate. A technical grade of mono-calcium phosphate was preventive apparently due to some inorganic contaminating material.

A preventive factor in wheat bran and middlings was reported by Lee (232), while Hunter, Dutcher and Knandel (180, 183) found that oats or oat feed (hulls, shorts and germ) and oat hulls were protective. Titus and Ginn (373) and Titus (371) reported that rice bran was preventive. This was confirmed by Milby (269). Branion (28, 29) reported that corn contained a factor which produced slipped tendons, a finding confirmed by Schaible (335) and Wilcke (417, 418), while wheat germ, oats, oat dust (shorts and germ) and oat feed were preventive. Sherwood and Couch (348, 349) concluded that even in rations having a satisfactory mineral balance, another factor is necessary in the ration if slipped tendons are to be prevented. This factor was present in wheat gray shorts and in rice bran in appreciable amounts; oat groats, cottonseed meal and peanut screenings contained lesser amounts. Henderson (158) found that egg yolk or chicken fat did not prevent the abnormality. Graham, Pettit, Sykes and Howell (119) attempted to extract the preventive factor from wheat germ. Schaible, Moore and Conolly (337) claimed that soyabean oil meal was preventive. Wilgus, Norris and Heuser (405) rated the perosis preventing properties of some feeds as wheat germ 100, wheat standard middlings 65, red dog flour 50, wheat bran 40, ground oats 30, wheat 5, and soyabean oil meal 0. Yellow corn and dehydrated alfalfa meal had a slight preventive action.

Sherwood and Couch (349) confirmed the preventive action of wheat gray shorts but found that rations containing 1.90 to 1.93% calcium and 0.87 to 0.99% phosphorus, even though containing 30% of this material, produced more slipped tendons than did rations containing 1.20 to 1.62% calcium and 0.56 to 0.57% phosphorus. Wilcke (402) reported that 10 to 20% of corn bran, 10% of ground oats, and 5% yeast were ineffective, whereas 10% of rice bran or 20% of oats prevented the condition.

Finally Sherwood and Fraps (350) announced that the factor in wheat gray shorts which prevented slipped tendons was not an organic substance but was one or more of the mineral constituents of the ash. Wilgus, Norris and Heuser (406) as a result of their findings with monocalcium phosphate almost simultaneously reported that perosis could be prevented by either 0.0035 or 0.0160% of manganese in the diet, and the perosis-preventing properties of feed stuffs were roughly related to their manganese content. A mixture containing 0.0025% each of manganese, aluminium and iron was preventive at lower calcium and phosphorus levels, while aluminium and zinc had a less effective action. Further results with

manganese were reported by Gallup and Norris (114, 115), indicating that manganese did not prevent all cases of perosis under certain conditions. Van der Hoorn, Branion and Graham (387) using purified diets encountered a leg abnormality, presumably a mild type of perosis, which was prevented by manganese. Milne (274) found silicon to be without influence. Heller and Penquite (156) found a water extract of rice bran to be preventive, although water extracts of wheat bran, wheat gray shorts, wheat embryo or alfalfa were not very effective. The ash of rice bran was preventive and the ash of wheat germ slightly so, although in an earlier paper (154) they found the ash of rice bran to be ineffective. They observed a correlation between the manganese content of the ration and perosis-preventing property, so they concluded that it may be the deficient factor. However since some of the best protective rations had no manganese addition, they concluded that other factors were involved. Miller and Bearse (419) encountered perosis on corn and barley diets but not in birds fed oats or wheat. The manganese content of the grains was oats 4.66 mgms. per 100 grams, wheat 2.91, barley 1.19, and corn 0.38 mgms.

Lyons, Insko and Martin (238) confirmed that protective action of manganese, but noted no protective action of aluminium nor zinc, rather the reverse, Since the mode of action of manganese in preventing perosis was not known, they eliminated its action in the digestive tract by an ingenious experiment, involving intraperitoneal injections. Such injections of the proper amounts of manganese were effective in preventing perosis, whereas zinc, aluminium or iron were valueless. Schaible, Bandemer and Davidson (336) found a wide variation in the manganese content of feeding stuffs and even a variation in the same feed from different sources. They confirmed the perosis-preventive action of manganese, but concluded that all sources of manganese, using various salts and ores, were not equally satisfactory.

Wiese, Elvehjem, Hart and Halpin (401) found that, although 10% of rice bran prevented slipped tendons, alcohol, glycerol or acid extracts of rice bran did not. Since autoclaving the bran destroyed a factor which prevented perosis, they concluded that there was some labile substance which either alone or in association with manganese was operative in preventing perosis on the particular ration used. The addition of 0.0025 and 0.005% of manganese to their simplified ration protected, whereas with another ration, containing higher levels of calcium and phosphorus, neither 15% of rice bran nor the addition of manganese was effective. They also observed some interesting difference in total blood phosphorus on these various diets. Schaible, Bandemer and Moore (336a) have studied some physico-chemical effects of excess minerals in the gut.

It is obvious from this mass of evidence that the perosis picture is not complete. It seems safe to conclude that excessive minerals, particularly phosphorus, are an etiological factor, although one cannot be positive, even in this regard, since the level of phosphorus in the ration and the incidence and severity of perosis do not go hand in hand. It seems obvious that manganese is *a* protective mineral, but it is also obvious that Mn *is not* the *only* protective factor involved. In view of the variation in the manganese content of feeding stuffs, the minute quantity required, and the fact, which will be considered later, that excess manganese is

harmful, it does not seem advisable nor sound practice to advocate, as has been done, the addition of this mineral to commercial poultry rations. If perosis develops on a ration, the addition of manganese to that particular ration, under the particular environmental conditions may be justified. Since the *modus operandi* of this mineral is not yet clear, its use under such conditions may not give the desired results.

Moreover, it seems evident that a histological study of the tibio-metatarsal joint and its attendant ligaments may be necessary to ensure that the various rations used by experimenters result in one and the same perosis.

5. POTASSIUM, SODIUM AND CHLORINE (SALT)

The alkali metals, sodium and potassium, are found normally throughout the body in the soft tissues and body fluids. Although they are closely related, their unequal distribution suggests selective separation and characteristic functions. Sodium, chiefly as chlorides, carbonates and phosphates, predominates in the blood and body fluids, whereas potassium occurs, chiefly as phosphates, to a greater extent in the cells, soft tissues and muscles. Although a deficiency of either sodium or potassium retards growth, their functions in the body are not clear. Potassium seems to be essential for proper cell formation. The ions of sodium, potassium and calcium, in proper balance, are concerned in the rythmical activity of the cell and are essential for heart activity. These alkali minerals are intimately connected with the regulation of the hydrogen ion concentration of the blood. Sodium chloride (salt) is the chief inorganic constituent of blood plasma, and presumably is the source of the chloride ion in the hydrochloric acid of the gastric juice. Salt metabolism is related to water balance and there is a relationship between sodium requirements and the adrenal glands.

Potassium, sodium and chlorine are excreted by the kidneys. Potassium is especially abundant in both plant and animal tissues, so no naturally occurring deficiency of this mineral should ever arise. On the other hand, farm rations, in general, particularly those consisting largely of cereals, do not contain sufficient amounts of sodium nor chlorine. Hence the need for common salt as a mineral supplement for livestock has been widely recognized. It is generally accepted that poultry rations should contain from 0.5 to 1.0% salt. However, such products as meat meal, fish meal and dried milk contain appreciable quantities of salt, and with poultry rations containing the usual amounts of these supplements, it is doubtful if 0.5% salt is required. However that amount does not appear to be harmful, under such conditions, and it is probably a safeguard, for a considerable variation in the salt content of feedstuffs does occur.

Mitchell and Carman (279) showed that chicks fed on a cereal diet, which was deficient in salt, evidenced retarded growth, with a decreased efficiency of food utilization, which was not due to a failure in digestibility. They concluded that a deficiency of sodium rather than of chlorine was involved. Prentice (322) reported that chicks fed on rations containing no salt made poor growth, with inefficient food utilization, marked susceptibility to disease and retarded sexual maturity. He also concluded that the beneficial effects of sodium chloride were due to sodium rather

than chlorine since sodium bicarbonate gave similar results while glycine hydrochloride was without effect. He recommended that a chick mash should contain 0.5% salt. Prentice suggested that grass plays an important part in balancing a ration deficient in sodium. Later, the same worker (323) showed that a lack of salt in the ration fed to laying hens resulted in a decreased rate of egg production and egg size, the birds losing weight and becoming subject to cannibalism. Mitchell and McClure (280) estimated that one egg contains 73 mgms. of sodium and 88 mgms. of chlorine, amounts furnished by 186 mgms. of salt. Common (71) has reported that calcium retention of laying birds is adversely affected by a lack of salt, probably sodium deficiency.

In the reviewer's laboratory, chicks could be successfully reared on a simplified diet, normally containing 4% of McCollum salt mixture 185, in which 2% of this mineral mixture was replaced by 2% of grass ash, or on diets in which the mineral mixture was entirely replaced by 3.5% grass ash and 0.5% salt. Complete substitution of grass ash for the salt mixture resulted in little growth, indicating that grass is deficient in either sodium or chlorine or both.

Some poultrymen consider that the use of salt increases the palatability of the ration, but Payne (307) could not confirm this.

Excessive amounts of salt are toxic to chickens. Collier (69) was able to feed 0.042 ounce per hen per day without any symptoms, but 0.063 ounce caused diarrhoea. He concluded that 1 ounce of salt per day could be fed safely to 100 mature birds. Wheeler (398) stated that a safe level was 5 ounces of salt to every 100 pounds of feed. Suffran (363) reported a case in which fowls were poisoned by being fed a mash made of potatoes to which salt had been added, chemical analysis showing that each bird had ingested 10 to 14 grams of salt. From subsequent experiments he concluded that a dose of 4 grams per kilogram body weight was lethal. Edwards (97) also reported a case in which fowls and ducks were killed by being fed the sweepings, containing about 22% salt, from a bakery. Using pigeons, he determined the minimum lethal dose as 3.33 to 4.50 grams of salt per kilo body weight. The symptoms noted by these workers were inability to stand, intense thirst and convulsions, with a viscous discharge from the beak. Post mortem examination showed hemorrhages and congestion in the gastro-intestinal tract. Other workers have reported lesions in many organs, particularly the kidneys. Heinz and Haas (150) also encountered salt poisoning among birds eating heavily salted garbage. Pearl, Surface and Curtis (311) reported a serious case of salt poisoning.

Mitchell, Card and Carman (275) found that chickens could be fed from 9 to 21 weeks of age, on rations containing as much as 8% of salt, with no detrimental effect on their condition, nor rate of growth, once the birds became accustomed to the ration. A daily intake of 6 to 8 grams exerted no harmful effects on birds 9 weeks or older. Salt given in solution twice daily was more toxic than equal amounts consumed in the feed. The minimum lethal dosage for birds weighing from 3 to 5 pounds was 4 grams per kilogram body weight (*i.e.* 0.4% of the body weight). Parker (303) arrived at a similar conclusion. Quigley and Waite (326) fed baby chicks on rations containing from 1 to 15% salt; 8% or greater levels resulted in less growth due to the unpalatability of the ration. They also

determined the minimum lethal single dose to be 4 grams per kilo body weight, although a larger dose could be tolerated if divided.

Torrey and Graham (375) reported that ducks are more susceptible to salt poisoning than chickens.

Sjollema (352) fed chicks on rations containing only 0.034 to 0.013% sodium, and obtained poor growth and food utilization in comparison to those receiving 0.5 and 1.0% salt. He found that 2% salt (0.8% sodium) in the ration was injurious. The sodium requirements of chicks were estimated as 0.4 to 0.5% at 1 month of age and at 0.30 to 0.35% at 2 months. Halpin, Holmes and Hart (131) reported that rations containing 1% salt resulted in somewhat better growth, when fed to baby chicks, than rations with no salt addition, and very much better growth than with rations containing 5% salt. In a later experiment (132) using baby chicks and pullets they concluded that 5% of salt was more than was desirable, that 2% of salt was not injurious, neither was it advantageous, obtaining satisfactory results with 1%.

Heller (151) reported that naturally alkaline and saline waters were injurious to chicks, but an adjustment to these drinking waters could be achieved, though at the expense of egg production. Selenium may have been partially responsible for the injurious effects.

Some poultrymen recommend the use of a "soda flush" (sodium bicarbonate) for chicks. Delaphane (83) and Witter (410) have shown that this is not a safe practice, since excess amounts or continued administration results in kidney damage.

6. IODINE

The hormone or regulatory agent secreted by the thyroid, two separate lobes laterally situated at the base of the neck, is known as thyroxin. It is a derivative of tyrosine, containing about 60% iodine. Most of the iodine in the body is found in the thyroid since it has a special affinity for iodine. The lobes are filled with a colloid material containing iodine in an organic combination with globulin, known as thyroglobulin. It is probable that thyroxin exists in the gland as thyroglobulin and that other iodine compounds may be present in the gland.

Iodine comes from the sea, and the soil and plants of inland regions are deficient in this mineral. In such areas simple, endemic, or colloid, and in some instances congenital goitre, an enlargement of the gland due to increase in the colloid material, arises as a result of this lack of iodine. The Great Lakes region of North America and part of the Prairie Provinces of Canada are such regions. The administration of iodine alleviates the condition.

Welch (396) in Montana, reported that "goitre in poultry is very common." "Flocks with a very high percentage of it have been noted in goiterous areas. The enlarged thyroid, frequently as large as the thumb, is entirely concealed by the feathers, and so far we have not observed any bad effect on the health of these fowls. The owners of goitered flocks have always reported that egg production and general health were normal. We have attempted to determine whether congenital goitre existed among newly hatched chicks but have been unable to get the material to study. No losses have been reported, at any rate, from this cause." Kernhamp (208)

gave a detailed report of a simple colloid goitre in poultry, and mentioned that in Minnesota, goitre in poultry is extremely uncommon.

The weight of the thyroid per kilo of body weight and the percentage of iodine therein, is greater in poultry than in mammals, according to Chaudhuri (62) and Cruickshank (80, 81). A higher metabolic rate results as shown by body temperature of 104 to 108° F., with more rapid respiration, heart beat, digestion and absorption and greater food consumption per unit weight than mammals. In view of these circumstances, it might be considered that goitre would be a fairly common poultry disease. However the disease appears to be almost non-existant. Several factors are operating in this connection. Marine products such as fish meal, kelp, oyster shells and fish oils, which all contain appreciable quantities of iodine, are common ingredients in poultry feeds. Moreover the use of iodized salt (ordinary salt containing 0.02% or more of potassium iodide) is common. In this connection the voluminous literature dealing with the benefits or lack of beneficial effects from supplemental iodine feeding is contradictory. Many workers have added iodine, usually as potassium iodide, to a basal diet but either did not determine the amount of iodine in this diet, or neglected it. This basal may or may not have contained adequate amounts of iodine, without any further supplementing. Moreover, even a proven benefit of iodine feeding in a goitre area is no proof that in non-goitre regions iodine supplements will be beneficial.

Chaudhuri (62) and Cruickshank (80, 81) have shown that there is a relative increase in the iodine content of the thyroid with the attainment of sexual maturity. Cruickshank (80, 81) has also shown that the iodine content of the thyroid is affected by ingestion of food, age, species and season. Iodine administered either orally or injected is rapidly removed by the gland. Orr and Leitch (298) found that the thyroids of birds receiving 1 mg. of iodine daily for three weeks contained 0.3 to 0.5% iodine (fresh weight basis) as compared with 0.1 to 0.2% for the controls. Simpson and Strand (351) obtained similar results. The thyroid weight and total iodine increased in proportion to body weight in the growing chick. There is a seasonal variation in thyroid weight (from 0.085 gm. in March to July to 0.13 gm. from January to March) and a similar variation in percentage and total iodine. This was related to egg production since the greatest weight was previous to the period of maximum egg production and the iodine content is depleted somewhat after a period of heavy egg production, as confirmed by MacOwan (266). Ovarian tissue contained only a trace of iodine, the small ova very small amounts, and the mature yolks appreciable quantities. Cruickshank (81) reported that Latimer of Minnesota (a goitre area) gave the average weight of the thyroid as 0.173 gm., as compared with 0.13 gm. obtained in England with the same breed, at the same period of the year. She concluded from a histological study that the thyroid was in relatively great activity in the immature bird and inactive in the laying and moulting hen.

That the thyroid gland is concerned in moulting and feather development is evident from the work of Cole and Reid (68), Crew and Huxley (79), Giacommi (117), Hutt (184, 185), Krizenecky (217, 218), Torrey and Horning (376, 377), Zavadovsky (412, 413), Cole and Hutt (67), and Prawochenski and Slizynski (321).

Hamilton and Kick (135) were unable to obtain any beneficial effects by the addition of iodine to a basal mash fed to young chickens. Similar findings were reported by Forbes, Karns, Bechdel, Williams, Keith, Callenbach and Murphy (108), and Holmes, Pigott and Packard (175). Conflicting results have also been obtained as to the value of iodine administration to laying birds, probably as has been pointed out, the amount of iodine in the control ration being the determinant factor. Negative findings have been reported by Forbes *et al.* (108) who could find no definite effect of the iodine on egg production. Similarly Malan (240) found that hens receiving an iodine supplement to a ration not considered to be deficient in iodine, showed no advantage in mortality, egg production or fertility. Wehner (395) reported that feeding 0.5 gm. iodine daily had no effect on the course of the moult, general health, egg production or hatchability. Similarly Johnson, Pilkey and Edson (194), although in a goitre area, found that supplementary iodine feeding did not improve egg production, fertility, hatchability, or mortality; and Lee, Hamilton and Henry (233) could demonstrate no improvement in egg production or mortality as a result of feeding either inorganic or organic iodine as a supplement to a laying mash containing 715 parts per billion of iodine originally. Almquist and Givens (5) have found no significant difference in egg production or hatchability. Kelly (204) reported that in one trial, egg size was increased by the addition of iodine to the diet of the hen, in another the reverse was true, while a third trial gave inconsistent results. Edson (96) found that the addition of iodine to a ration containing 19 to 41 parts per billion gave no improvement in egg size, percentage of thick albumen, yolk index, weight or colour, or shell thickness.

On the other hand, Simpson and Strand (351) increased the egg production of old hens, who also appeared healthier with more rapid moulting and recovery. Similar conclusions were made by Klein (213), Scharrer and Schropp (333), and Zaitschek (or Zajtay) (411). These latter workers also reported increased hatchability. Crew (78) reported that senile females were markedly stimulated to egg production by large doses of thyroid, although Cole and Hutt (67) found that 59 mg. of dessicated thyroid per pound of live weight had no appreciable effect on egg production.

Mitchell and McClure (280) estimate the daily iodine requirement of a five pound chicken as 4.5 to 9.0 gamma (a gamma is 0.000001 of a gram, or 1 microgram), and Orr and Leitch (298) give the requirement of a 4-pound fowl as 5 gamma. The use of the usually recommended amounts of iodized salt would adequately meet these requirements. Although poultry can withstand considerable amounts of iodine, according to Orr, Crichton and Middleton (297), Schmidt (338), and Scharrer and Schropp (333), being more resistant than mammals, there is danger of overdosage with iodine. Schmidt (338) found that doses of 0.3 to 0.5 grams reduced the size of eggs, and 1.5 gm. interrupted the laying period. Decreased egg size was encountered by Peano and Pissaro (309). Brown (31) reported that hens refused to eat a mash containing 0.2% potassium iodide. Zavadovshii, Lipichina and Radsivon (414) and Hutt (185) showed that daily doses of 4 mg. thyroid iodine per 1 to 2 kilograms of body weight was lethal. Asmundson, Almquist and Klose (7) fed iodine in amounts varying from 800 to 16,000 gamma daily to hens fed on a ration containing 50 gamma

per 100 gm. Iodine was fed in various forms as oyster shell, dessicated thyroid, sodium iodide, potassium iodide, iodo-salicylic acid (which was not readily absorbed), di-iodotyrosine and iodized olive oil for 20 weeks. The iodine content of the eggs varied from 3 to 1730 gamma per egg, but body weight, egg production and feed consumption were not affected adversely, except in the case of those birds fed thyroid and those receiving the high dose of sodium iodide.

The percentage of iodine in the egg can be increased many times by the addition of either inorganic or organic iodine to the ration of the hen, as shown by Ambrosio (6a), Simpson and Strand (351), Jaschik and Kieselbach (192), Straub (361), Chrzaszcz (63), Peano and Pissaro (309), Zickgraf (415), Viollier and Iselin (390), and Sumita, Kawabata and Fujioko (364). Wilder, Bethke and Record (403) concluded that the iodine content of hens' eggs varies directly with the amount of this element in the ration, but Almquist and Givens (5) reported that the iodine content of eggs was not closely proportional to the iodine content of the ration. The iodine content of eggs has been measured by Cole, Curtis and Bone (66), while Vezzani, Devalle, Meynier and Simonetti-Guizza (389) have used iodized eggs successfully in the treatment of goitre. The bromine content of eggs can also be increased, Purjesz, Berkessy and Gonczi (324, 325), and Berkessy and Gonczi (12, 13).

In our laboratory, the results of a co-operative experiment with the Toronto Hospital for Sick Children, showed that the incorporating of an organic form of iodine in a rachitic diet did not lessen the vitamin D requirement of chicks nor did it increase the efficiency of irradiated ergosterol in promoting calcification.

Free iodine in the form of colloidal iodine has been employed as a disinfectant, Chandler (60).

7. IRON AND COPPER

Hemoglobin, the red colouring material in blood, consists of a protein globin and heme, as iron-containing pigment. This compound is responsible for most of the oxygen carrying capacity of blood. Iron is essential for the formation of hemoglobin; in its absence nutritional anaemia results. In addition to the iron in hemoglobin, which is the largest fraction, a smaller amount is present in muscles as myoglobin, some as tissue iron and reserves of varying level in the spleen, liver and kidney. Iron is probably present in every living cell and seems to be concerned in vital activities, particularly physiological oxidations, being a constituent of several respiratory enzymes such as cytochrome.

It seems to be generally agreed that iron to be absorbed must be soluble, ionisable and ultrafilterable. Reduced iron, ferric hydroxide and salts soluble in acid solutions meet these requirements. This mineral is almost wholly excreted by the intestine.

The utilization of iron in the rebuilding of hemoglobin (hematopoietic process) depends upon traces of copper. The addition of pure iron increases the iron content of the liver and spleen but only in the presence of copper is this stored iron used in the hematopoietic process. Commercial iron salts are usually contaminated with sufficient copper. Elvehjem, Hart

and Kemmerer (98) have shown that both iron and copper are required for hemoglobin synthesis in chicks.

The green leaves of young plants are rich in iron, and other feeds with a good iron content are meat products, legume seeds, cereal grains and cane molasses.

The hemoglobin level of chicks has been reported as between 6 and 7 gm. per 100 cc. by Hart, Elvehjem, Kemmerer and Halpin (140), and as 8.2 gm. for male and 7.5 for female chicks by Cook and Harmon (76). Holmes, Pigott and Campbell (172) followed the hemoglobin level of chicks from hatching to 21 days. The hemoglobin level at hatching was high, being 10.8 gms. per 100 cc. on the average, as would be expected from the results of Sendju (221) who found that the hemoglobin content of incubated eggs increased slowly to 22 mgs. at the fourteenth day and rapidly to 140 mgms. at 20 days. McFarlane and Milne (263) and Szejnman-Rozenberg (368) have investigated the iron and copper metabolism of the developing embryo. Copper metabolism has been studied by Loeschke (236a) and Sumegi (363a). Following hatching, the hemoglobin level falls, reaching a low level of 8.8 to 9.0 gms. at 5 to 7 days. This period of "physiological anaemia" lasts until about the seventeenth day of life, at which time, the hemoglobin level begins to increase, being 9.6 gms. per 100 cc. at 21 days. However Holmes, Pigott and Campbell (171) found a wide range in hemoglobin values for 21-day old chicks, though they were from a common source and fed on the same ration. On the other hand, Hart, Elvehjem, Kemmerer and Halpin (140) concluded that a practical baby chick ration supplies enough iron and copper to prevent anaemia.

There is no doubt that anaemia is a cause of embryonic mortality during incubation, under certain conditions. Smith and Branion (355) and Smith (353, 354) encountered anaemic embryos in eggs laid by hens fed certain diets, particularly those containing tankage. Direct summer sunshine and/or good grass range alleviated this mortality. McFarlane, Fulmer and Jukes (262) could not correlate this condition with the iron and copper content of the eggs. Cavers and Hutt (59) reported a similar condition, which was more frequent in the fall and winter months, very few anaemic embryos being found in March or April.

In the laying hen, the demand for iron and copper for egg formation is large, since the average egg contains about 1.1 mgm. of iron and 0.067 mgm. copper. Elvehjem, Kemmerer, Hart and Halpin (99) give the values for egg yolk as 0.0143% iron, and the amount of copper as 0.00076% in yolk and 0.00056% in egg white. They were unable to increase these values by feeding extra iron and copper to the hen. Similar results were reported by Hoffman (164) and by Erikson, Boyden, Martin and Insko (103), although they found that hens with access to sunshine and blue grass range produced eggs with higher iron and copper content. This finding is probably significant, since anaemic embryos also tend to disappear, under similar conditions. Vecchi (388) claimed that the iron content of eggs could be raised by adding iron to the hen's diet. Although McFarlane, Fulmer and Jukes (262) encountered significant variations in the iron content of egg yolk, they could not correlate these variations with the ration. The copper content of eggs was extremely variable.

The data available on the hemoglobin content of the blood of mature birds is variable, the reported normal values ranging from 7.0 to 11.1 mgms. hemoglobin per 100 cc. It should be noted that some workers report the iron content rather than hemoglobin content of the blood. Among others Burmester (52), Scott, Serfontein and Sieling (344), Hayden and Fish (149), Dukes and Schwartze (91) Gonzaga (118), and Dyer and Roe (94) have reported hemoglobin or iron values. Some of the variation is due to technique, Schultze and Elevhjem (342); some to differences in the age of the birds, Dukes, Schwartze and Brandt (92); to the season of the year, since these last workers have shown that the hemoglobin level is higher in winter; to individual variation, Maughan (258) and Cook and Harmon (76) and possibly to the rate of egg production. It seems quite definitely established that the blood of chickens has a lower hemoglobin content than that of man, Burnett (53), Klieneberger and Carl (214), Forkner (109) and Maughan (258), and that males have a higher hemoglobin content than females, with capons intermediate. Some of these variations may be due to the fact that the hen's spleen serves as a reservoir for red cells, thus helping to regulate the hemoglobin level in the blood, Harmon, Ogden and Cook (138). This splenic reserve was found by Harmon (137) in cockerels, capons, pullets and laying hens but not in cocks nor broody hens.

Some mineral supplements containing iron have been sold as supplements to laying mashes to prevent nutritional anaemia in the hen. However, the evidence is not unanimous that anaemia accompanies egg production. Dukes, Schwartze and Brandt (92) found no correlation between spring egg production and the hemoglobin content of the blood of the laying hens. Winter (409) also concluded that production, confinement or moult do not produce quickly noticeable or marked changes in the iron content of the blood, although earlier (408) he stated that egg production does cause a decrease in the hemoglobin level of the blood. Schultze, Elvehjem, Hart and Halpin (343) found that a practical laying ration furnishing about 14 mg. of iron and 0.5 mg. of copper per hen per day could support heavy egg production without a resultant lowering of the hemoglobin content of the blood. Feeding of additional iron and copper failed to raise either the hemoglobin level or egg production. On the other hand, Maughan (258) and Cook and Harmon (76) observed that the hemoglobin level fell with high egg production, the fall occurring previous to the onset of egg production, when the yolks are forming. Feeding of additional iron increased the hemoglobin level in one case, and was useless in the other. Harmon (137) reported that the hemoglobin level was high at hatching and at maturity, but low at two weeks of age. Egg production distinctly decreased the hemoglobin level, but high producers had higher levels than low producers, when both were either in or out of production. The hemoglobin level rose rapidly with the onset of broodiness.

From this contradictory evidence, one is faced with certain possibilities. A judicious balancing of the laying ration may adequately meet the increased requirements for iron and copper for egg production. If not, especially if iron supplements may not be effective, then it is possible that the hen withdraws iron from her body stores in preference to dietary iron for deposition in the egg. If paleness in the head and comb can be

considered as one indication of anaemia, then many poultrymen would suspect that anaemia does arise in flocks laying heavily during the winter months, particularly in view of the fact that the hemoglobin level of newly hatched chicks varies. That this is not due directly to confinement, would seem evident from the results of Winter (409) and Doyle, Matthews and Roberts (90). Moreover, anaemic embryos are more common during the winter months, and although it seems evident that iron supplements do not increase the iron content of the egg, three facts need consideration. First, grass and sunshine apparently do raise the iron content of the egg; second, grass and sunshine or sunshine alone, prevent anaemic embryos; third, laying hens allowed to range on a good "short grass" range soon "redden up." One is tempted to theorize that ultraviolet light has some influence on hemoglobin (cf., Maughan, 257) and that young grass contains iron that is abundantly available, be that due to the form of the iron, the combination in which it is present, or other mineral compounds also contained therein. Iron metabolism in poultry nutrition would seem to be a still fertile field for research.

Marza, Marza and Chiosa (253) have made a study of the distribution of iron in the ovary of the hen.

Although many texts accept egg yolk as one of the best foods for hemoglobin regeneration because of its high iron content, Whipple and Robscheit-Robins (400) have shown that it is relatively inert for hemoglobin production. Sherman, Elvehjem and Hart (346) reported similar findings, since incomplete hemoglobin regeneration resulted from feeding egg yolk as a source of iron, together with 0.05 mg. copper, although the α-α-dipyridine method showed the iron to be nearly 100% available. The addition of 1 mg. of supplemental copper to the egg yolk gave normal hemoglobin regeneration, indicating that the failure was due to retarded utilization of copper, probably as a result of the formation of insoluble copper sulphide in the digestive tract. Summerfeldt (365), pointing out that the solubility of iron depends upon the degree of acidity present in the gastro-intestinal tract, and also on protein digestion which liberates iron bound with protein, made use of an artificial gastric juice, which simulated normal conditions found in the stomach of children. The solubility of the iron in egg yolk under these conditions was only 21%.

Deobald and Elvehjem (85) reported that the addition of large amounts of soluble iron or aluminium salts (0.9% Fe, 0.44% Al) to a normal ration, resulted in severe rickets, due to the formation of insoluble phosphates during digestion. These results may account for the beneficial results reported for iron and aluminium salts on perosis. Some of the excess phosphorus in the diet would be rendered unavailable to the chick.

8. MANGANESE

The relationship of manganese to perosis has already been considered. However, it is becoming more evident that this element plays other rôles in poultry nutrition. It has been reported as being concerned in hemoglobin regeneration.

Van der Hoorn, Branion and Graham (387) showed that it was an essential mineral for poultry. Keenan, Kline, Elvehjem, Hart and Halpin (420) modified the salt mixture used in a purified diet for chicks so as to

contain 0.246 gm. manganese sulphate, 0.209 gm. zinc chloride and 0.225 gm. copper sulphate per kilo, but they gave no reasons for this modification. Gallup and Norris (115) found that about 50 parts of manganese per million parts of the ration were necessary for growth, a reduction to 10 p.p.m. resulting in retardation. Similar findings were reported by Schaible, Bandemer and Davidson (336). The former workers also reported that pullets fed on a ration containing 13 p.p.m. manganese produced less eggs, with lower hatchability, than those fed a diet with 200 p.p.m. Increasing the manganese content of the hens' ration increases the manganese content of the eggs according to Vecchi (388). Gallup and Norris (115), and Lyons and Insko (237) who were able to increase the manganese content of the eggs laid by hens receiving a ration with 5.5 p.p.m. from 0.43 to 0.56 p.p.m. (dry matter) to 0.84 to 0.96 p.p.m. with 45.5 p.p.m. in the diet. Peterson and Skinner (312) gave the manganese content of egg yolk as 2.3 and McHargue (265) as 1.5 p.p.m. Bertrand and Medigreceanu (17) gave the content of the edible portion (yolk and albumin) as 2.52 and Peterson and Skinner (312) as 1.1 parts per million. Schaible, Bandemer and Davidson (336) gave the manganese content (mg. per kilogram) of some feedstuffs as follows: corn 4.1 to 7.8, wheat 24 to 37, wheat germ 155 to 167, oats 31 to 44, oat hulls 275, rice 19, barley 12 to 18, meat meal 4 to 5 and fish meal 16 to 93. They also investigated the availability of the mineral in the form of various salts, such as sulphate, dioxide, permanganate, carbonate, and ores such as rhodonite and rhodochrosite. All were not equally satisfactory.

One of the most interesting discoveries was that of Lyons and Insko (237) that hens fed on a low-manganese ration had low hatchability, with a high percentage of chondrodystrophic embryos. Supplementing the ration with manganese, iron and zinc resulted in good hatchability, even the embryos which died having normal skeletons. Chondrodystrophic embryos contained an average of 2.4 micrograms of manganese while those embryos with normal skeletal development contained 7.0 micrograms. That manganese was the element concerned was shown by injecting manganese directly into the egg, which prevented the abnormality. Zinc was ineffective.

This discovery is so interesting that it warrants further discussion. Chondrodystrophic embryos are characterized by "very short, thickened legs, short wings, parrot beak, globular contour of head, protruding abdomen and retarded down and body growth." Oedema is often present. This abnormality was first reported by Landauer and Dunn (227). Hutt and Greenwood (187) found that its frequency was high in January but declined steadily to a complete absence in June. Munro (283) also observed this seasonal incidence. Byerly, Titus, Ellis (55) concluded that vegetable proteins in the diet of the hen increased its incidence, but they did not observe any seasonal variation, nor did Upp (383). Later, Byerly, Titus and Ellis (56) reported a different type of chondrodystrophy which could be prevented by wheat germ and liver or wheat germ and whey in the ration or by allowing the hens access to direct sunlight and green grass. The findings of Smith (353, 354) and Smith and Branion (355) would confirm this. Landauer (226) differentiated between two types of this abnormality—sporadic and nutritional; the former has a peak of mortality

during the second week of incubation, the latter during the last few days. The sporadic type have bent tibiae, whereas the tibiae of the nutritional type are reasonably straight. Green grass presumably is a good source of manganese, as is wheat germ, and their preventive action could be thus explained. The rôle of sunshine is not clear.

It should be pointed out again, that van der Hoorn, Branion and Graham (387), Heller and Penquite (156) and Gallup and Norris (115) have shown that excess manganese is detrimental.

Manganese is eliminated in the faeces and urine.

Although the reviewer does not feel, as has been pointed out, that sufficient information is available to justify the addition of manganese to all poultry rations, nevertheless there undoubtedly will be some individuals who will desire to make such additions. Fifty parts per million of manganese would seem to be about the correct amount in the rations. One-quarter of a pound of a technical grade of manganese sulphate (containing 32 to 33% manganese) per ton of mash will raise the content well above the marginal level. It will be necessary to pre-mix this amount with some other ingredient in order to obtain uniform distribution in the completed mash.

9. MAGNESIUM

Magnesium is closely related to calcium metabolism. It is essential for certain body activities, being present in muscle tissues where it acts as a co-enzyme for the breakdown of adenylphosphate during muscle contraction. It is also a co-enzyme for phosphatase. About two-thirds of the magnesium is present in the skeleton, chiefly as carbonate, egg shell containing about 1.4%. It is excreted to some extent in the faeces and in the urine. In considering magnesium the distinction between food magnesium and blood magnesium is striking, since several disorders are characterized by hypomagnesaemia but not by magnesium deficiency in the ration.

As has been pointed out, dolomitic limestone often contains a sufficient percentage of magnesium carbonate to be harmful when used as a mineral supplement. Wheeler (399) showed that rations deficient in calcium but carrying abundant magnesium produced a shortage of calcium and mineral in the bones of chickens. He also showed that magnesium would not substitute for calcium in egg shell formation. Halpin and Hayes (130) reported greater egg production with oyster shell than with limestone which contained a relatively high percentage of magnesium. Buckner, Martin and Insko (40) found that the addition of magnesium carbonate to chick rations, in amounts sufficient to bring the magnesium level from 0.76 to 7.05%, disturbed the calcium and phosphorus balance, resulting in deformed bones with a low ash and calcium content. They also reported that in the earlier work at the Kentucky station they used limestones containing from 1 to 30% magnesium carbonate. Best results in bone formation and egg production were obtained when the percentage of magnesium carbonate in the limestone was small. A decrease in bone ash as a result of feeding 3% magnesium carbonate (0.89% Mg.) was encountered by Milby (268, 269) although growth was normal. Mussehl, Hill, Blish, and Ackerson (291) reported similar results and Schaible, Moore and

Conolly (337) obtained perosis with magnesium carbonate additions. Miller and Bearse (272) encountered difficulties with a phosphatic limestone, analysing 1.3% magnesium. Fluorine may also have been involved. Alder (4) found dolomite to be injurious. On the other hand Hart, Steenbock and Morrison (145) claimed that the addition of 4 or 5% of limestone containing as much as 40% magnesium carbonate promoted normal growth and reproduction in chickens.

However, it would seem safer to avoid the use of dolomite limestones in poultry rations. Suitable limestones should contain about 98% calcium carbonate.

Magnesium may be concerned in egg production, since Charles and Hogben (61) found the level of serum Mg to be as 2.08 to 2.71 mg. per 100 cc. in immature pullets, 1.87 to 2.87 mg. in laying pullets with no egg in the oviduct, and as 1.86 to 5.90 mg. with the egg in various stages of shell formation.

10. FLUORINE

Although fluorine is widely distributed in soils, rocks, waters and in plant and animal materials, it apparently has no function to perform in metabolism, in fact there is ample evidence that this element has detrimental physiological effects. However, because of its widespread distribution, and particularly because of its occurrence in rock phosphate and phosphatic limestone sometimes used in mineral supplements, fluorine requires consideration in this review.

Drinking water containing less than 1 p.p.m. of fluorine appears to be harmless, that containing 1 to 2 p.p.m. is harmful, and concentrations over 2 p.p.m. decidedly so. Phosphatic rocks have a variation in fluorine content from about 0.4 to 4.25%. Apparently the toxicity of various fluorine salts is not the same, since the element in phosphorite minerals, although presumably fluoapatite, $3\ Ca_3(PO4)_2Ca\ F_2$, is from 10 to 20 times as toxic as the fluorine in calcium fluoride. Chickens seem to be less susceptible to fluorine toxicosis than larger farm animals.

Wheeler (397) concluded that steamed bone meal was superior to rock phosphate for growing chicks. Similar results were obtained by Hartwell and Kirkpatrick (146) and Hartwell and Lichtenthaeler (147). Kennard and White (207) found that rock phosphate could replace steamed bone meal in a mineral mixture for laying hens. However Buckner, Martin and Peter (42) found that rock phosphate caused diarrhoea and decreased egg production. Kistler (211) also reported that bone was superior to raw rock phosphate in the production of eggs. Buckner, Martin and Insko (38) reported that 5% of rock phosphate in a chick ration depressed growth and increased mortality. They concluded that rock phosphate was only injurious during the early life of the chick.

From a study of the effect of rock phosphate, containing 3.52% fluorine, on the growth of chicks, Halpin and Lamb (133) found that 1% of this supplement had no effect, 2% resulted in some depression of growth, and 3% was decidedly harmful, although the ash content of the bones was normal. The two lower levels had no influence, but the high level depressed egg production without affecting hatchability.

Kick, Bethke and Record (209) concluded that chicks could tolerate more fluorine in the ration as calcium fluoride than as rock phosphate or sodium fluoride. The fluorine of rock phosphate and of sodium fluoride were similar in action. Toxic levels of fluorine resulted in decreased growth and food consumption, but did not affect the percentage of bone ash. The clotting time of the blood was decreased. A review of fluorine in animal nutrition, including poultry, was given by Kick *et al* (209a).

The Ohio workers set the toxic level at 0.10% fluorine or more and recommended that not more than 1% of rock phosphate should be fed.

Similar results were reported by Hauck, Steenbock, Lowe and Halpin (148) feeding from 0.015 to 1.2% of sodium fluoride. Levels up to 0.15% were ineffective, but greater amounts were toxic, depressing appetite and growth, the high levels lowering serum calcium. No significant effect on bone ash, kidney phosphatase or on the parathyroids resulted. Hens fed 0.96% sodium fluoride lost weight and egg production was depressed. The toxicity of fluorine was enhanced by thyroid feeding, according to Phillips, English and Hart (313).

Phillips, Halpin and Hart (314) showed that the addition of fluorine as rock phosphate to the ration of laying hens resulted in a distinct increase in the fluorine content of the egg. Haman, Phillips and Halpin (134) reported that practically all normal tissues of the hen contain fluorine, bones having the highest concentration and the more active tissues lower amounts. The addition of 0.1056% of fluorine, either as rock phosphate or phosphatic limestone raised the fluorine content of bones 13 to 14 times and from 2 to 3 times the normal in the soft tissues.

It seems obvious that it is not a safe practice to feed rock phosphate or phosphatic limestone as mineral supplements to poultry.

11. SELENIUM

This element is present in a number of plants growing upon certain soils, in concentrations ranging from mere traces to 0.2%. Feeding of such plants or their products results in "alkali disease" or selenium poisoning. A conservative estimate is that any plant product containing 5 p.p.m. selenium is potentially dangerous. The element in the vegetation is derived from the soil due to iron pyrites and other sulphide ores containing selenium, chiefly in shales of the Cretaceous period. Any soil containing 0.5 p.p.m. or upwards, especially in regions of low rainfall is potentially dangerous. Water from such regions may contain 2 to 3 p.p.m. of selenium. It has also been found in rock phosphates.

Tully and Franke (379) have reported that chicks fed on a ration containing 65% of seleniferous grains showed impaired growth, ruffled feathers, nervousness and delayed and reduced egg production; 25% of affected grains was harmless. The eggs produced had low hatchability, almost three-quarters of the eggs which failed to hatch having deformed embryos or monsters, Franke and Tully (111, 112). Similar teratoes were produced by injecting selenium salts into eggs before incubation, Franke, Moxon, Poley and Tully (110). Poley, Moxon and Franke (320) found that a balanced ration containing grains, whose selenium content was 15.15 p.p.m., fed to laying hens caused a loss in weight, due probably to

decreased food consumption, with a decrease in egg size but egg production and ferility were unaffected. In seven days, the hatchability was reduced to zero, but hatchability returned to normal, six days after removal of the seleniferous grains.

Poley and Moxon (319) fed laying rations containing 2.5, 5, and 10 p.p.m. of selenium. With the intermediate level the hatchability was not appreciably affected, although some chicks had wiry down. The high level reduced hatchability to 0, the dead embryos showing short upper beak, oedema of head and neck, missing toes and eyes and wiry down, characteristics of selenium poisoning. No effects on body weight, egg production or fertility were noted. Moxon and Poley (282) investigated the selenium distribution in the body of these hens. This was increased proportionally to the amount in the ration. The selenium content of the eggs was also raised.

12. SULPHUR

Sulphur is chiefly found in the body in combination with proteins, being a constituent of the essential amino acids cystine and methionine. It is present in considerable amounts in the albuminoids such as are found in the hairs, feathers, scales, nails and beak. Only organic sulphur appears to be absorbed so there is no nutritive advantage in adding inorganic sulphur to rations.

In addition to its structural functions, it plays a rôle in cell oxidations by virtue of its presence in glutathione. It is also a constituent of vitamin B_1.

Herrick and Holmes (161) have found that the feeding of sulphur will largely prevent caecal coccidiosis in chicks, if fed in sufficient amount. On the other hand, Holmes, Deobald and Herrick (167) reported that feeding flowers of sulphur to chicks for six weeks, even with cod liver oil, resulted in rickets. Takashi (368a) produced typical calcium metastases and decalcification of the bones of pigeons by feeding sulphur or sulphur-containing compounds.

Marlow and King (251) concluded that the sulphur in egg yolk and white was organically bound and identical, and that nearly all the sulphur in eggs can be accounted for as cystine and methionine sulphur.

Mohler (280a) reported that feeding flowers of sulphur or inorganic sulphur compounds, as done by some poultrymen, had no influence on the moulting period.

13. BERYLLIUM

The reviewer in co-operation with the Toronto Hospital for Sick Children investigated the effect of beryllium carbonate additions to normal chick rations. One-half per cent had little or no effect but 1 and 2% levels reduced the inorganic blood phosphorus to 2.5 and 1.6 mgs., and the bone ash to 36.0 and 32.4% respectively. This effect is due to the formation of insoluble beryllium phosphate in the gut. However the chick seems to be somewhat resistant, since Guyatt, Kay and Branion (123) produced this effect with 0.125% beryllium carbonate in rats. Duncan and Miller (93) were unable to produce this syndrome by feeding up to 50% of soil containing 0.223% beryllium, to chicks.

14. OTHER MINERALS

Although such minerals as zinc and cobalt have been shown to have special functions in mammals, there does not seem to be any proof existant as to their value for chickens. Silicon is a constituent of feathers, but there is no indication that it is ever a limiting factor for feather production. However, it is becoming exceedingly evident that these rarer minerals, even though present in minute amounts, may be extremely important nutritive elements. The rôle of "trace" minerals in poultry nutrition would appear to be a field worthy of further investigation.

In addition to the specto-graphic analysis previously given, Bertrand and Agulhon (16) have reported the presence of boron in eggs. Underhill, Peterman, Gross and Krause (382) gave the aluminium content of eggs as 0.017 mg. per 100 gms. Bishop (25, 26) and Taylor (369) reported the presence of lead in eggs, Mankin (248) that of molybdenum; and Hubbell and Mendel (177) the presence of zinc in eggs, and Bertrand and Vladesco (18) in eggs and the organs of poultry. Straub and Donck (361a) have also reported on the mineral constituents of eggs.

Van der Hoorn, Branion and Graham (386) encountered a new leg deformity, tentatively termed "arthritis" by the use of casein purified by acetic acid leaching in a purified diet. That a mineral or minerals were concerned was indicated by the fact that the inclusion of the ash, made by concentrating and ashing the leachings from the casein, prevented this abnormality. Later results showed that grass ash was preventive also. The factors involved did not appear to be any of the better known minerals including manganese.

15. BLEACHING FUNCTION OF MINERALS—OTHER EFFECTS

Maw (259) has found that bone char has a definite bleaching effect on the colour of fat produced during the fattening period, as well as on the fat already in the body. This action was better than bleaching agents such as fuller's earth and nut charcoal. This finding is particularly important in the fattening of birds for markets which demand a white fat in dressed poultry.

Card and Sloan (57a) could find no effect of mineral supplements on interior egg quality. Hendricks (158a) reported that mineral supplements had no influence on length of wing or tail feathers.

16. EFFECT OF MINERALS ON RESISTANCE TO DISEASE

Clapham (64) has shown that chicks receiving a diet deficient in calcium and phosphorus were less resistant to infestation with *Heterakis gallinae* (caecal worms) than those receiving adequate amounts of these minerals. This is not surprising, since it is generally agreed that a dietary deficiency in any essential constituent lowers the body resistance to disease. On the other hand, Holmes, Herrick and Ott (168) reported that excessive mineral additions were detrimental to chicks infected with *Eimeria tenella* (coccidia). In view of the suggestion previously made, that under modern feeding conditions poultry are more liable to suffer from an excess rather than deficiency of minerals, this finding is noteworthy. The reviewer is tempted to suggest, with little or no proof, that the indiscriminant use of

mineral supplements to poultry rations is not helping, to say the least, to overcome intestinal infestations in poultry. It does seem feasible to point out that it should be within the realm of possibility to make the intestinal tract of growing chicks an unappealing home for parasites. Although the work of Heller and Penquite (155) and Mussehl, Blish and Ackerson (290) indicate that the digestive tract is able to adapt itself to wide variations of dietary conditions without any distinct modification in pH, the author still desires to indulge in theory.

Although the range of the Department of Poultry Husbandry at Guelph has been in continuous use for over 30 years, there has been a decided lowering of mortality in the past few years, due primarily to a decrease in intestinal disorders. Several factors have operated in this connection, and any of these or a combination of all, may have been responsible. First, a continuous supply of fresh succulent green feed is provided throughout the season by the judicial choice of crops and "strip" planting. The colony houses are moved from strip to strip, as the green feed is ready. This involves of course, the ploughing and cultivation of the soil throughout the season, and sunshine and aeration of the soil are valuable "disinfectants". Succulent green feed is mildly laxative and presumably keeps the digestive tract in good working order. Secondly, coarse mashes are used in place of finely ground mashes. This may also serve as a stimulus to the digestive tract. Thirdly, the mineral supplements, bone meal, oyster shell and insoluble grit are fed in separate hoppers, the chickens being allowed to do their own mixing. Under these conditions, disease has been reduced, rather than increased as would be expected from the continuous use of "old" range.

To ensure a good, fast growing crop, the ground is heavily fertilized each year with a complete fertilizer, at the rate of 200 to 300 pounds per acre. English workers (193) have reported that large amounts of fertilizers could be picked up from the soil by birds without affecting health.

17. GRIT

Presumably the function of grit is to aid in the grinding of food and to prevent impaction in the gizzard, but in reality its function is not known. Ever since Spallanzani (357) remarked "we may reasonably conclude that the collecting of pebbles is less the effect of choice than stupidity", the question of the function of grit, its value if any, and the kind to be used, has been debated.

The function of grit was investigated by Mangold (241, 242, 243), by Mangold and Feldin (245), Mangold and Kath (246) and by Mangold and Rudiger (247) who concluded that the presence of small stones or grit in the gizzard allows poultry to derive more benefit from grain. Klee (212) also believed that grit was necessary to maintain birds in good health. Jull (196) also found that grit was necessary for the economical digestion of whole grain. Halnan (128) considered that the efficiency of the gizzard for grinding was increased by grit, and Kruger (219) believed that the presence of grit increased digestion. Although Jaeckel (191) found that the presence or absence of grit did not change the power of contraction of the gizzard, its presence kept birds in better physical condition. This problem has also been studied by Katayama (199), Kato (200), Danilova,

Solun, Chlebnikov and Poliakov (82a), and Ferber and Bruggeman (107). The function of the gizzard has been summarized by Mangold (244) and Henry, Macdonald and Magee (159).

On the other hand, Buckner and Martin (34) and Kaupp and Ivey (202) were unable to demonstrate any benefit from the use of grit, or harm as a result of its absence. Moreover, Buckner, Martin and Peter (48) and Bethke and Kennard (19) have been able to rear chicks on a gritless ration. Burrows (54) has shown that the gizzard itself is not essential to life.

Fritz, Burrows and Titus (113) have found that gizzardectomized birds digested coarse feeds much less efficiently, whereas with finely ground feeds, there was little difference. Bird, Oleson, Elvehjem, Hart and Halpin (24) encountered an abnormal thickening or swelling of the gizzard lining in chicks fed a fine, gritless ration, with a trend towards retarded growth.

Limestone is often sold as grit. Limestone and oyster shell are calcium supplements which dissolve in the digestive tract, particularly in the gizzard under the action of hydrochloric acid. It probably takes from 24 to 48 hours for these substances to dissolve and in the meantime they can serve as grinding agents. On the other hand, grits like granite or quartz are insoluble, and, although they break up by shattering, they probably remain for much longer periods in the gizzard than do soluble grits. Platt and Stephenson (316) reported that limestone grit remained in the gizzard as long as mica grit. Waite (392) considered that limestone or crushed oyster shell could serve in the dual rôle of a calcium supplement and of grinding material.

Although grit is not essential, it seems safe to conclude that its use is a wise practice, for it enables the bird to more efficiently grind up the coarser feed particles, thus releasing the nutrients bound therein. Moreover, if one is willing to grant that grinding is the function of grit, then durability—hardness and insolubility—is the criterion for judging its value. Such insoluble grits as quartz, feldspar, granite and mica are to be preferred. It is unfortunate that limestone is termed grit. If the name grit was reserved solely for insoluble products, then no confusion would arise. Poultrymen who wished to feed "grit" for its grinding value would not be "misled" into purchasing a calcium supplement. Those who wish to feed a "dual-purpose" supplement could do so also.

18. COMPLEX OR SIMPLE MINERAL MIXTURES?

There has been considerable discussion on the relative merits of complex mineral mixtures, made up of many ingredients, as against simple mixtures consisting of such supplements as bone meal, oyster shell or limestone, salt (usually iodized) and occasionally iron. From a survey of experimental evidence, and a consideration of practical poultry experience, it seems evident that simple mixtures work reasonably well. Kennard, Holder and White (206) found that a mixture of 60 parts bone ash, 20 parts calcium carbonate (limestone or oyster shell) and 20 parts of sodium chloride (salt) fed at a 2% level or less, gave satisfactory results. Hart, Steenbock, Halpin and Johnson (143) secured good results from a mixture of 5 parts of raw bone (approximately 50% calcium phosphate), 5 parts of pearl

grits (supplying calcium carbonate), and 1 part of salt. Orr, Moir, Newbigin, Robertson and Murphy (300) and Orr, Moir, Kinross and Robertson. (299) used a mixture of 50% bone meal, 20 of common salt, 20 of chalk, 5 of sulphur, 5 of iron oxide, and a trace of iodine. Phillips and Hauge (315) recommended a mixture of 24 parts of limestone, 15 parts of salt, and either 21 parts of soluble bone, 22 parts of steamed bone, or 42 parts of acid phosphate. We have obtained excellent results with bone meal, oyster shell and iodized salt.

However, until such time as the exact amount of all the rarer minerals required by poultry is known, and until the time when complete soil and crop analyses for all such minerals are known, if ever, one cannot condemn the use of complex mineral mixtures, if the purchaser consideres them economic and provided they are sold as *mineral supplements, no more, no less.* It is quite within the bounds of reason that, under certain climatic and soil conditions, some of the "extra" minerals contained in these minerals may be beneficial. On the other hand, they may be of no value. Only a carefully controlled feeding test would settle that question, and even such a test, conducted in one locality, would not be applicable to all other localities. However, even when a poultryman has obtained benefits from a complex mineral mixture, he must also satisfy himself that such benefits were not due to some of the "common" supplements such as bone meal or limestone, in such a mixture. If they are, then he is not economically justified in paying the higher price for the complex supplement.

The moment such complex mineral mixtures are sold as a "cure-all" or preventive for all poultry disease, they are no longer mineral supplements but are drugs, and as such have no place in everyday poultry feeding. Furthermore, it should be clearly borne in mind that no mineral mixture will ever become the panacea for all poultry ailments.

19. PRACTICAL CONSIDERATIONS

In view of the varying amounts of such protein supplements as meat meal, fish meal or dried milk, and also to the varying mineral content of these products, it is impossible to recommend any definite amount of a mineral or various minerals which should be fed. For this reason, the reviewer strongly advocates the feeding of such supplements as bone meal, oyster shell or limestone and insoluble grit in separate hoppers for both chicks and laying hens and allowing them to partake of such supplements as they desire. Experience has shown that this is a safe practice.

The grains and their products are decidedly deficient in calcium, but usually they furnish adequate amounts of phosphorus. When supplements like meat meal, fish meal or dried milk, in the generally recommended amounts are fed, they usually correct the calcium deficiency, except in the case of laying hens. The calcium requirement of laying hens for the production of egg shell is so high that it is imperative to feed some calcium supplement such as oyster shell. It may happen that rapidly growing chicks may require additional calcium and/or phosphorus, under some conditions, but from $\frac{1}{2}$ to 1% of bone meal and limestone would meet any such requirement. The poultryman should be warned again that even such amounts often will result in perosis. However, if vegetable protein

supplements such as soyabean oil meal are used in chick rations, replacing part of the meat meal or fish meal, the resultant deficiency in minerals, usually calcium, must be compensated by adding 1% of bone meal or calcium carbonate for each 3 to 5% of the "animal" protein replaced.

The amount of salt required will depend also upon the kind of protein supplement used, but the addition of ½ to 1% of salt to mashes either for chicks or laying hens appears to be safe. In inland regions the use of the same amount of iodized salt would seem to be justified.

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FIELD STUDIES OF ALFALFA INOCULATION[1]

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The problem of securing successful inoculation of alfalfa is often perplexing when first undertaken. Partial failure frequently tends to the opinion that the crop is poorly suited to the conditions. From 1915 onwards many puzzling situations were encountered at the Beaverlodge Substation and each in turn has contributed its share of interest.

INOCULATION EXPERIMENTS

The 1928 Seeding

In 1928 at the suggestion of the Dominion Bacteriologist, a seeding of alfalfa was made at Beaverlodge to investigate the effect of inoculation at varying intervals in advance of seeding. The soil-seed method was employed, the soil being obtained on the station premises. The land was a loose black loam. By July the following year readily apparent differences in colour and height occurred in each of the four sets. Plots inoculated immediately in advance of seeding were no better than the uninoculated checks. Inoculation 24 hours in advance of seeding was more successful. Decidedly the best results were obtained from plots inoculated a week in advance of sowing, while plots sown with seed inoculated two weeks in advance about equalled those sown with seed inoculated the day before seeding.

Colour and other growth contrasts were lost by the following season but yields corresponded to those of the first crop. In the third crop the effects continued in a diminishing degree.

In the three-year period the plots sown with seed inoculated immediately before sowing outyielded the checks by only 1.5%. The plots inoculated 24 hours in advance of sowing outyielded the checks by 14.1%, those inoculated one week in advance by 22.3%, while those inoculated two weeks in advance yielded 14.9% more than the checks.

The 1930 Seeding

The scope of the work was amplified in 1930 when a seeding was made on the Johnson farm adjoining the town of Beaverlodge. Both culture and soil-seed inoculation were used. Seedlings were made on three dates commencing May 21. The soil was then mellow and slightly dry. June rains puddled the soil, which was a medium grade dark clay loam, and subsequently shepherd's purse threatened the stand so much that the first two seedings were mown on July 26 and August 4. Other weeds were hand-pulled. Some winter-killing of the alfalfa occurred where the weed growth had been the thickest.

By midsummer of 1931, contrasts began to appear but differing in nature from those of the 1928 seeding. In the 1930 seeding no sharp

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FIGURE 1. Alfalfa, 1930 seeding. Left, culture inoculation; right, soil inoculation.

differences could be discerned as between prompt or deferred seeding after inoculation, but there was an unvarying advantage of soil- over culture-inoculation. This was greatest in the second cropping season (1932) when the check plots were outyielded 130 to 160% by the soil-inoculated plots and 25 to 56% by the nitro-culture plots. The contrasts were at first the most marked on the loamiest areas. On other areas they still existed in the third cropping season.

In one respect the results of the 1930 seeding agreed with those of the former trial. Such contrasts as occurred were slow in manifesting themselves and proved persistent.

Undoubtedly weed growth and soil puddling depressed the yields materially, but it is not entirely clear why plants which were known to have developed nodules in the year of seeding took two or three years to develop the characteristics of well-inoculated plants.

The 1932 Seeding

In 1932 field tests were made on a gray podsol soil on the farm of Clarke Bros., northeast of the station. Soil-seed and culture inoculation were again employed and the seed was sown immediately after inoculation, 24 hours after inoculation and one week after inoculation, respectively. Two cultures were used, one from Ottawa (No. 173), and another, said to be acid-tolerant, received from Dr. H. G. Thornton, of Rothamsted, England. Both cultures were sown with and without calcium di-acid phosphate. The seed was sown in a dry period and very little growth

was made at first. By the end of the season stands were thin, the colour was pale, and no contrasts were evident.

By July 15, 1933, some definite response was noted in the Rothamsted-culture plots. The one sown with seed inoculated by this culture a week in advance of seeding showed distinctly deeper colour and some extra vigour of growth. The 24-hour inoculation showed somewhat less and the immediate inoculation less still, though its response was readily discernible. At this date plots inoculated with the Ottawa No. 173 culture gave no definite indications that the treatment had been successful. The addition of phosphate was without effect.

The contrasts extended and deepened in 1934. The Rothamsted-culture plots again grew much the tallest and continued the deepest green. The Ottawa No. 173 plots darkened considerably, though they did not show as much evidence of strong inoculation as the former. The soil-inoculated plots remained decidedly yellowish in appearance, with almost as little colour as the checks. Still no benefit appeared from the use of the phosphate.

The interval of inoculation in advance of seeding seemed to have no pronounced effect. Stands were somewhat patchy and growth was uneven due to the unfavourable conditions in the year of seeding and to ice killing during the winter of 1933–34.

The 1934 Seeding

A further seeding was made June 12, 1934, on the Clarke farm under favourable conditions. The following inoculants were employed: Ottawa

FIGURE 2. Alfalfa 1934 seeding. Left, inoculated with Ottawa mixed culture; center, not inoculated; right, soil inoculation.

mixed culture; Ottawa No. 173 culture; Beaverlodge No. 3 culture (isolated from nodulated plants grown at Beaverlodge); Rothamsted acid-tolerant culture; soil (from the Beaverlodge substation); check (no inoculation).

Each of these was compared in three intervals of inoculation in advance of seeding, *viz.*, inoculation one week in advance; inoculation 24 hours in advance, and inoculation immediately in advance. The soil was dry when the seed was applied but the sky was overcast and a heavy shower fell within a few hours.

On August 28 the Ottawa mixed culture, the Beaverlodge No. 3 and the Rothamsted cultures seemed to be distinctly the most effective. The checks were rather pale in colour, while the soil-inoculated plots showed occasional green plants. Ottawa No. 173 proved slightly better than the soil but hardly equal to the other three inoculants.

In 1935 the Rothamsted-culture ranked first at the beginning of the season, closely followed by the Ottawa mixed and the Beaverlodge No. 3 strains. During the six-weeks' period between the first and last observations there was a gradual narrowing of these differences so that by July 17 there was little to choose in appearance among these inoculants. The soil-seed plots ranked next in order and improved their comparative position as the season advanced. The Ottawa No. 173 and the check plots took fifth and sixth places, respectively, both ranking much below the other four treatments. They failed to improve as the season progressed.

Although conditions seemed reasonably uniform at time of seeding, the plot arrangement seemed best adapted to a study of the effect of inoculants rather than of the results of pre-inoculation periods.

Yields agreed pretty well with the field notes. On the average of six plots representing a duplicate test with each of three inoculation pre-intervals the Rothamsted culture produced 3771 pounds of hay per acre; the Ottawa mixed 3660 pounds; soil-seed, 3289 pounds; Beaverlodge No. 3, 3133 pounds; Ottawa No. 173, 2668 pounds; and the checks, 1946 pounds, notwithstanding some accidental inoculation of the latter.

By September 25 the variation that had been so apparent throughout the season's first growth was hardly noticeable in the aftermath. Infestations which had before been limited to the margins were now general over most of the area. The extreme wetness of the summer undoubtedly favoured bacterial diffusion.

FINDINGS OF OTHER WORKERS

The effect of holding inoculated seed has been studied by a number of workers (1, 3, 5, 6, 7, 8). Although there is an immediate decrease in the number of organisms adhering to the seed coats, viable bacteria may be retained after six months or more, capable of producing nodules. Tests at Ottawa (5) have shown that the nodule-forming capacity tends to decrease with time of holding, though the decline is less rapid than that of the organisms themselves. It was also found that with lower temperature of storage the nodule forming capacity decreases to a less extent than at higher temperatures.

In a field trial, following inoculation four days in advance of sowing, Fred, Whiting and Hastings (4) found about one-fourth of the plants

vigorous, with the remainder small and lacking nodules, and attributed this to the death of bacteria before reaching the soil. On the other hand Thornton (8) found that although storing of inoculated seed for 28 days halved the number of nodules, there was no significant reduction in yield.

Thornton stresses the importance of proper soil aeration, high moisture content, and the action of phosphates, which stimulate nodulation and root development. From tests in different parts of England he concludes that it is likely the spreading of bacteria is affected by season, and occurs only under suitable conditions of soil moisture and temperature. The soil reaction is important, particularly in its bearing on the adaptation of bacteria. Some strains may form nodules at much lower pH ranges than others, suggesting that in some cases there may be more acid-tolerant strains in the soil than found in cultures usually supplied. Alway and Nesom (2) found that in some land soil spreading gave much better inoculation than pure cultures and that a second sowing of alfalfa succeeded well after the first sowing had failed.

DISCUSSION OF THE BEAVERLODGE RESULTS

Since in commercial practice it is occasionally necessary to hold inoculated legume seed some days before sowing, the 1928 seeding had a practical basis. Prepared culture was not available at the time and the soil-seed method of inoculation was resorted to. The results obtained cannot be entirely explained, but suggest that the bacteria on moistened seed may have gone through a period of incubation following the inoculation and that this continued, at least as long as the soil remained moist.

The 1930 seeding was likewise on dark soil where the chance of previous inoculation was slight. Here adverse conditions affected the stand, prohibiting fine comparisons, but it was significant to observe the paired contrasts of the results of culture and soil inoculation, with the darker colourings spreading first from the loamiest areas. The bacteria transferred with the soil in each test must have included strains better adapted than those supplied by the culture, yet it is not clear why it required in some instances at least three seasons to achieve its full result and why in places the inoculated plots gave scarcely better results than the checks.

The introduction of the Beaverlodge No. 3, Ottawa mixed, and the Rothamsted strain to the Clarke farm, in contrast to the Ottawa No. 173 strain, may serve to explain the relatively poor showing made by the culture series in the 1930 seeding. In view of this it would seem to be necessary to determine the adaptive range of any strain of nodule bacteria before prescribing it for use, and would suggest the greater safety of using a composite of several strains until the superiority of any one was definitely established. Peace River soils are far from uniform. Mostly they are podsolized and tend to be slightly acid in reaction. In such a case a culture of mixed strains might be safest in general practice.

SUMMARY

The results of six years testing under field conditions confirm the findings of other investigators that there are pronounced differences in the vigour, adaptability, and beneficial effects of various stocks of nodule bacteria, and suggest the differentiation of these for specific conditions.

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Soil-seed inoculation applied by the glue method gave much better results than culture inoculation in some seedings but was not uniformly better. Lacking a handy supply of a suitable strain of culture, soil-seed inoculation may be used to advantage but prepared culture would seem to be preferable, if only in minimizing the danger of weed-seed and disease transference.

Inoculation of legume seed several days in advance of sowing may be satisfactory, but this may depend somewhat on storage conditions, and possibly other factors.

The addition of calcium di-acid phosphate to the seed when the culture was used produced neither positive nor negative results.

Ample moisture and good cultural conditions are favourable to the securing and spreading of effective inoculation.

Artificial inoculation may be done according to rule without proving effective. Under some conditions it may not become fully effective for two or three years.

The grey woodland soils will produce satisfactory stands of alfalfa under suitable management.

On land not previously inoculated no cultural or bacterial treatment can be used with guaranteed success. If the first attempt results in failure the sensible course is to try again on the same gound, taking advantage of the bacteria introduced and multiplied by the first attempt.

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THE RELATION OF WIREWORMS TO POTATO GROWING IN THE PRAIRIE PROVINCES[1]

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Wireworms cause important losses annually in the majority of gardens and potato fields in large sections of Western Canada. This is especially true in the prairie area and over a broad margin of the adjacent park belt, where it applies to all but the heaviest soils. In the latter, as well as in the densely wooded country and in much of the irrigated land of Alberta, the damage is less severe and less common although occasionally of serious proportions. In general, observations indicate that as with small grain crops, the rate of wireworm damage is considerably greater to potatoes which have been planted on land that was summer-fallowed the previous season.

The major damage to potatoes is in the loss of market value due to disfiguration of the tubers, but sometimes the yields are also materially reduced. In recent years the damage has increased, reaching such magnitude in 1936 that should it continue on this scale, it may force the abandonment, in some sections, of commercial production of potatoes. For example, in the Saskatoon district in 1936, fields up to 75 acres in area suffered as much as 50% reduction in the yield of marketable tubers. In considerable measure, this can be attributed to an increase in the abundance of the pest probably connected with the general neglect of weeds in fallow land during the recent depression period. In part it is undoubtedly connected with the direct influence of drouth conditions upon feeding habits, and it is to be expected that the injury will be materially less during seasons of abundant moisture at critical periods. However, the problem is really a "permanent" one because the characteristics of the chief species, the prairie grain wireworm (*Ludius aeripennis destructor* Brown), eminently fit it to be a major pest of potatoes in this region, the net result being that the problem is most acute in the areas and fields which in all other respects are best suited to the production of potatoes.

Because potatoes are usually planted in old grain fields, the wireworm problem of potato growing is inseparably linked with the wireworm problem of grain production. Consequently, extensive field studies on the latter have provided an essential background from which to approach the problem with respect to potatoes. Such studies have been supplemented by observations in wireworm-infested potato fields and by preliminary experiments with baits and soil fumigants. Investigation of this problem is continuing, our knowledge in several important directions being definitely inadequate.

SUMMARY OF CONTROL RECOMMENDATIONS

The solution of the problem (1) depends upon either *avoiding* severely infested soil through careful selection of fields, or sufficiently *reducing* the

[1] Presented before the Horticultural group of the 17th Annual Convention of the Canadian Society of Technical Agriculturists at Saskatoon, Saskatchewan, June 1937.

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heavier infestations before the planting of potatoes. The latter can usually be accomplished gradually, but cheaply, by frequent clean summer-fallowing, and sometimes by means of baits; or quickly, but at considerable cost, by the use of crude naphthalene as a soil fumigant.

Choice of Land

Selecting land "safe" for potatoes is often feasible because some of the main factors, such as soil type and cultural practices, which largely determine wireworm abundance, are known.

There is a high probability of freedom from serious wireworm damage in the following types of fields: where good clean summer-fallowing has been practised about every three years, or oftener; in heavy clay soil, except where recently broken from native or tame grass sod; in the more dense park areas, land which has been under clean cultivation for from four to ten years; and in irrigated sections, fields of medium or heavy soil which have been irrigated at least three times annually for several years.

On the other hand, there is a high probability of serious wireworm damage occurring to potatoes planted in the following types of fields: those which have remained "idle" for several years; where, as a rule, weeds have been poorly controlled during the summer-fallow years; where summer-fallowing has been infrequent, three or more crops being grown in succession; where grass has been grown continuously for several seasons; and in willow-scrub land which has been under cultivation for less than three years.

In the severely-infested districts, special care should be taken to avoid fields with unfavourable histories, even though at the time of planting they appear attractive as potato land. A valuable "indicator" of the importance of the wireworm infestation in a field is the amount of damage occurring to young wheat plants of the first crop after summer-fallow. Such observations should be made on fields which are being considered for future use in potato production. Wherever appreciable thinning by wireworms occurs to wheat on fallow, potatoes are likely to be severely damaged. The symptoms of wireworm injury to wheat are characteristic and easily recognizable (4).

Summer-fallowing

From the standpoint of wireworm control (3, 4), the essential measures in summer-fallowing seem to be, keeping the field absolutely free of all large weeds from the middle of June to at least the end of July, presumably to starve the newly-hatched wireworms which are then seeking their first food; thorough shallow tillage during the last half of July to destroy a portion of the pupae which are found at that time in the top three or four inches of soil; and ensuring that all tillage is never deeper nor more frequent than is necessary for the control of weeds, frequent deep tillage appearing to favour the survival of the pest.

Two such summer-fallowings, three years apart, have been known to reduce a severe wireworm population by fully one-half, and there is evidence that summer-fallowing in alternate years, or in two or three successive years, would still more quickly reduce infestations.

As long as proper summer-fallowing continues to hold an essential place in any sound permanent farming program in the Canadian Prairies,

the above procedure has the advantage of being not only persistently effective in controlling wireworms, but also highly economical in that it involves no additional costs but only practical modification of normal procedure.

Baiting with Potatoes

As an alternative method, for small areas, baiting with potatoes has been found to have some value in reducing wireworm numbers. Unfortunately, the effectiveness of this method is variable, depending upon several factors which are not yet fully understood though it is evident that the moisture factor is of great importance. But the method is cheap and applicable during crop years prior to the regular planting as well as when the land lies fallow. Preliminary tests on both lightly and severely infested fields have shown that, during persistently dry weather, single baitings frequently fail to trap more than 5% of the wireworms in the baited area. On the other hand, excellent control by this method has been obtained[4]. In this instance the authors observed that the main catch was secured during a period when a large proportion of the wire worms had moved into a superficial layer of moist soil, after a good rain which followed a considerable period of dry weather.

Soil Fumigation

Fumigation with crude naphthalene is recommended for land where the immediate and virtually complete destruction of wireworms is of sufficient economic value to justify a rather heavy outlay for materials and labour. Just how long such land would remain free from economic infestations would depend largely on the farming (or irrigation) practices subsequently adopted; but, in dry land farming with proper summer-fallowing at least once every three years, it is believed that the pest could be held below serious proportions for several, or even many, years. The present evidence is that reinfestation of the prairie grain wireworm by either migration or multiplication is very slow.

The use of crude naphthalene under dry land conditions in the Prairie Provinces (2) is a modification of the method developed by Lane (4) for the irrigated sections in the state of Washington. Successful use of this material appears to depend upon the following principles of operation:—

1. The application of the naphthalene when the soil temperature is high enough to ensure both larval activity and rapid formation of effective concentrations of gas;
2. The ploughing of the material into the soil, deeply enough, usually 7 inches in Saskatchewan, to permit placing a portion of it as deep as the wireworms are likely to be at that season;
3. The thorough working of the naphthalene through the soil, immediately after ploughing; and
4. The use of enough naphthalene to give lethal concentration of vapour throughout the wireworm-infested soil layer.

[4] Mr. H. J. Kemp, cerealist at the Dominion Experimental Station, Swift Current, Sask., reports that, in 1930 and 1931, through the use of potato baits, a heavily infested area was rendered suitable for cereal variety test purposes, as many as thirteen wireworms being taken at a single bait piece. This plot area of one and one-half acres is reported to be still free from serious wireworm numbers.

Lane recommends that the soil be 70° F. or higher at 6 inches below the surface. Thus, in the Prairie Provinces, the use of the method is generally limited to midsummer and to fallow fields. He also regards the thorough working of the soil after treatment as a very important point since naphthalene vapour apparently does not spread far in the soil with killing effect. For this reason two thorough and deep operations of a one-way disc or a duckfoot cultivator may be preferable, although one such operation has been known to give good results.

The application of eight hundred pounds per acre of approximately *95% pure naphthalene* is recommended by Lane for the treatment of a 12-inch depth of moist soil. This rate should be ample for use under any conditions on the Canadian Prairie. In fact, the preliminary tests (2) indicate that half this rate may here be adequate, at least in very dry soil and under our usual conditions of infestation which require treatment only to a depth of about 7 inches. Even at the lower rate, the cost of materials, at current prices, is some twenty dollars ($20) per acre; to which, of course, must be added the considerable expense of the thorough deep tillage that is required.

Field tests with calcium cyanide under Saskatchewan conditions in 1934 and 1935 failed to give satisafctory control of wireworms. Over 200 pounds per acre of "Cyanogas-G Fumigant" was drilled-in adjacent to rows (18 inches apart) of living wheat seedlings which served as bait to concentrate the wireworms. The material was applied both with a regular farm seed drill and with a hand seeder, these being the only seeding implements normally available to farmers in this area. As a consequence of the poor results secured and the excessive cost of the materials, it is evident that calcium cyanide cannot be recommended for wireworm control in the Prairie Provinces.

SUMMARY

Wireworms cause important losses annually to potatoes in the Canadian Prairies, especially in those areas and fields which in all other respects are best suited to the production of this crop. During the recent drought years both the infestations and the rate of damage have increased, reaching such proportions in some districts as to threaten the abandonment of potato production on a commercial scale. The solution of the problem depends upon either avoiding severely infested soil through proper choice of land, or reducing the heavier infestations, either gradually and cheaply, by frequent clean summer-fallowing (which may be supplemented, in small areas, by the use of baits), or rapidly but with increased expense by fumigation with crude naphthalene which has been found to give effective control under dry-farming conditions.

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BOOK REVIEW

SMITH, KENNETH.—A Text Book of Plant Virus Diseases. J. & A. Churchill Ltd., 104 Gloucester Place, Portland Square, London.

The *Textbook of Plant Virus Diseases* by Dr. Kenneth Smith of Cambridge University, England, (J. & A. Churchill Ltd., 1937) is a timely addition to the literature of plant pathology. Owing to the multiplicity of papers that have been published during recent years describing plant viruses and virus diseases, plant pathologists have found it difficult to decide whether viruses discovered locally have not been studied and described already elsewhere. Smith's textbook brings together all the principal descriptions of plant viruses and virus diseases in a systematic manner based upon the virus nomenclature scheme suggested by Dr. James Johnson of Wisconsin at the last International Botanical Conference in Amsterdam. The viruses are given the generic name of a host and arabic numerals to indicate distinct types and letters in addition where distinct strains of a major type are described. This method preserves the continuity of current practice, *e.g.*, "Nicotiana virus 1A" and "1B" represent two strains of "Johnson's Tobacco virus 1." The generic name chosen represents the host upon which the effect of the viruses was first described or upon which subsequent descriptions of virus symptoms were more adequate. Owing to the support already given this scheme of nomenclature by both American and European workers, the publication of this textbook will tend to establish order in the chaotic condition of the virus literature with respect to nomenclature.

Very few descriptions of plant viruses have escaped review in chapters I to VII inclusive. Dr. Smith is fully aware that he has given arabic numbers to signify distinctiveness to viruses that later may be proven to be closely related strains of the same basic type. Already, in spite of the limitations of serological methods in virus classification, relationships have been established by this method between plant viruses formerly considered distinct.

The description of the insects involved in virus transmission and their life histories in chapter VIII serves to focus attention upon insects as vectors of virus diseases, a field that is a challenge to scientists. In spite of the highly infectious character of "Solanum Virus 1" as judged by plant juice transfers and the fact that the majority of European and practically all American varieties of potatoes are infected, no one has secured satisfactory evidence that this virus is transmitted from plant to plant by thrips or other insects.

The final chapter deals with a large group of plant species that exhibit chlorotic or other symptoms that suggest virus infection but wherein the infective character of the disease has not been proven.

A three-column appendix of 37 pages is included to assist students in the diagnosis of virus diseases. All common hosts are listed alphabetically opposite brief descriptions of the diseases to which they are susceptible and the names of the viruses involved.

W. NEWTON.

ERRATUM

In the December 1937 issue, Volume XVIII, No. 4, the illustration over the caption for Figure 19, page 192, belongs to Figure 20, page 195, and the illustration over the caption for Figure 20 belongs to the caption for Figure 19.